ACCESS DB # 182175

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Scientific and Technical Information Center

SEARCH REQUEST FORM

Requester's Full Name: Susanna Moore Examiner # : 82304 Date: 3/14/2006

Art Unit: 1624 Phone Number: 2-9046 Serial Number: 10/560,386

Location (Bldg/Room#): Rem 5831 (Mailbox #): Rem 5C71 Results Format Preferred (circle): PAPER DISK

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To ensure an efficient and quality search, please attach a copy of the cover sheet, claims, and abstract or fill out the following: MPEP

Title of Invention: _____

Inventors (please provide full names): _____

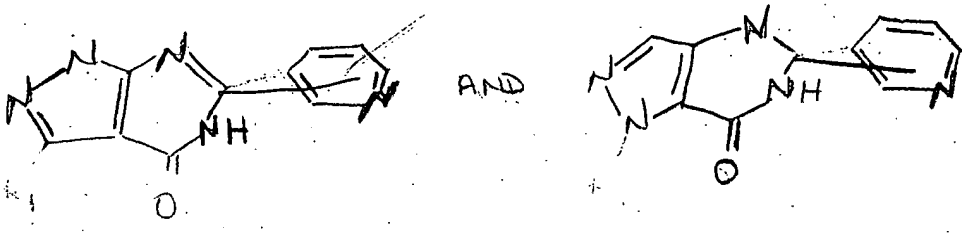
_____

Earliest Priority Date: _____

Search Topic:

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc., if known.

For Sequence Searches Only *Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*



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STAFF USE ONLY	Type of Search	Vendors and cost where applicable
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Searcher Phone #: _____	____ AA Sequence (#)	____ Questel/Orbit ____ Lexis/Nexis
Searcher Location: _____	____ Structure (#)	____ Westlaw ____ WWW/Internet
Date Searcher Picked Up: _____	____ Bibliographic	____ In-house sequence systems
Date Completed: _____	____ Litigation.	____ Commercial ____ Oligomer ____ Score/Length ____ Interference ____ SPDI ____ Encode/Tran: ____ Other (specify)
Searcher Prep & Review Time: _____	____ Fulltext.	
Online Time: _____	____ Other	

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=> d his ful

     (FILE 'HCAPLUS' ENTERED AT 14:29:12 ON 16 MAR 2006)

     FILE 'REGISTRY' ENTERED AT 14:48:55 ON 16 MAR 2006
L12             STR
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L15          46 SEA SSS FUL L12 OR L13
L16             STR
L17          44 SEA SUB=L15 SSS FUL L16

     FILE 'HCAPLUS' ENTERED AT 15:05:31 ON 16 MAR 2006
L18           4 SEA ABB=ON  PLU=ON  L17
                D STAT QUE L18
                D IBIB ABS HITSTR L18 1-4
L19          47 SEA ABB=ON  PLU=ON  "INOUE HIDEKAZU"/AU OR "INOUE HIDEKAZU C O
                DAINIPPON S"/AU
L20         608 SEA ABB=ON  PLU=ON  INOUE H/AU
L21          14 SEA ABB=ON  PLU=ON  ("MURAFUJI H"/AU OR "MURAFUJI HIDENOBU"/AU)
L22           1 SEA ABB=ON  PLU=ON  "HAYASHI YASHIHIRO"/AU
L23         728 SEA ABB=ON  PLU=ON  "HAYASHI Y"/AU
L24          56 SEA ABB=ON  PLU=ON  (L19 OR L21 OR L22) NOT L18
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L25           1 SEA ABB=ON  PLU=ON  (L20 AND L23) NOT (L18 OR L24)
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L28           3 SEA ABB=ON  PLU=ON  ((L20 OR L23) AND (PDE OR PHOSPHODIESTERASE
                 OR PYRIDINYLPYRA?)) NOT (L18 OR L24 OR L25)
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     FILE REGISTRY
     Property values tagged with IC are from the ZIC/VINITI data file
     provided by InfoChem.

     STRUCTURE FILE UPDATES:   14 MAR 2006  HIGHEST RN 876856-38-1
     DICTIONARY FILE UPDATES:  14 MAR 2006  HIGHEST RN 876856-38-1

     New CAS Information Use Policies, enter HELP USAGETERMS for details.

     TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

       Please note that search-term pricing does apply when
       conducting SmartSELECT searches.

     ************************************************************
     *                                                          *
     * The CA roles and document type information have been removed from *
     * the IDE default display format and the ED field has been added,   *
     * effective March 20, 2005.  A new display format, IDERL, is now    *
     * available and contains the CA role and document type information. *
     *                                                          *
     ************************************************************

     Structure search iteration limits have been increased.  See HELP SLIMITS
     for details.

     REGISTRY includes numerically searchable data for experimental and
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predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

FILE HCAPLUS

FILE COVERS 1907 - 16 Mar 2006 VOL 144 ISS 12
FILE LAST UPDATED: 15 Mar 2006 (20060315/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> fil hcaplus
FILE 'HCAPLUS' ENTERED AT 15:05:31 ON 16 MAR 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE COVERS 1907 - 16 Mar 2006  VOL 144 ISS 12
FILE LAST UPDATED: 15 Mar 2006  (20060315/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
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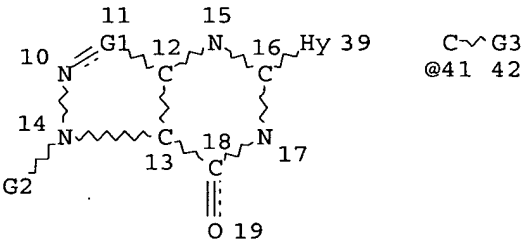


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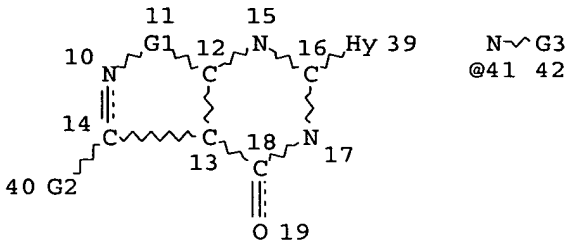

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STEREO ATTRIBUTES: NONE
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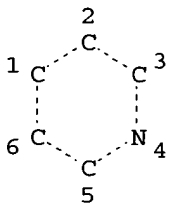
L16 STR



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L18 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L17

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L18 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:1127384 HCAPLUS
DOCUMENT NUMBER: 142:74598
TITLE: Preparation of (pyridinyl)pyrazolopyrimidinone derivatives as PDE 7 inhibitors
INVENTOR(S): Inoue, Hidekazu; Murafuji, Hidenobu; Hayashi, Yasuharu

PATENT ASSIGNEE(S): Daiichi Suntory Pharma Co.,ltd., Japan; Daiichi Suntory Biomedical Research Co.,ltd.
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004111054	A1	20041223	WO 2004-JP8643	20040611

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 2003-170094 A 20030613
OTHER SOURCE(S): MARPAT 142:74598
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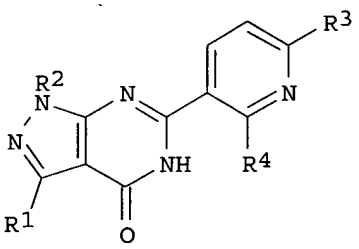

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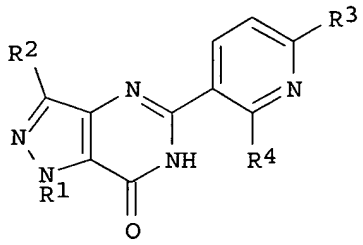

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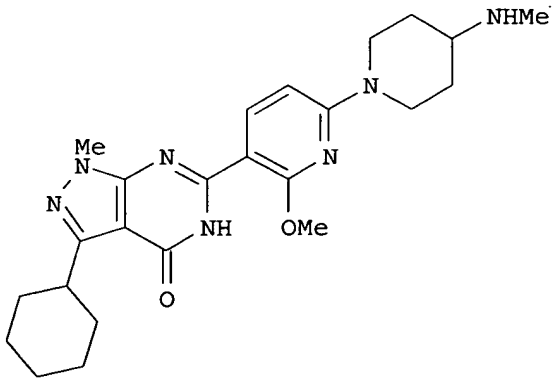


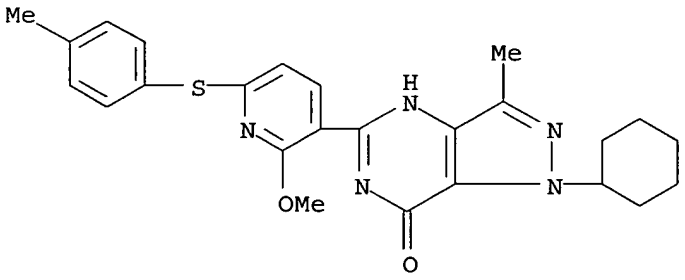
III

AB Title compds. represented by the formula I & II [wherein R1 = (un)substituted cycloalkyl or CMe3; R2 = H or alkyl; R3 = amino, COR7, SO0-2R8; R4 = H or (un)substituted alkoxy; R7 = alkoxy or amino; R8 = H,

halo, amino, (un)substituted alkyl, aryl; and pharmaceutically acceptable salts or solvates thereof] were prepared as PDE 7 inhibitors. For example, III was given in a multi-step synthesis starting from Me 2-methoxy-6-(4-methylphenylthio)pyridine-3-carboxylate. III showed inhibition of PDE 7 inhibitors with IC50 values of 0.0026 μM. Thus, I & II and their pharmaceutical compns. are useful for the treatment of various kinds of disease, such as allergic disease, inflammatory disease or immunol. disease (no data).

IT **812650-17-2P 812650-18-3P 812650-19-4P**
812650-20-7P 812650-28-5P 812650-29-6P
812650-33-2P 812650-36-5P 812650-39-8P
812650-40-1P 812650-46-7P 812650-47-8P
812650-50-3P 812650-51-4P 812650-52-5P
812650-53-6P 812650-54-7P
RL: PAC (Pharmacological activity); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(preparation of pyridinyl pyrazolo[3,4-d]pyrimidin-4-ones and pyrazolo[4,3-d]pyrimidin-7-ones as PDE 7 inhibitors)

RN 812650-17-2 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[2-methoxy-6-[(4-methylphenyl)thio]-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



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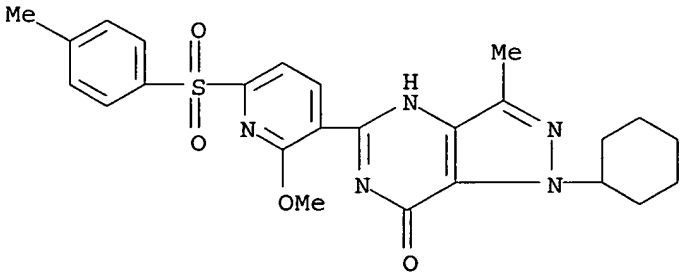
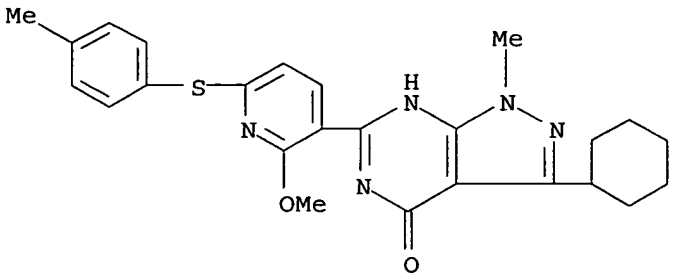


RN 812650-19-4 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-6-[2-methoxy-6-[(4-methylphenyl)thio]-3-pyridinyl]-1-methyl- (9CI) (CA INDEX NAME)



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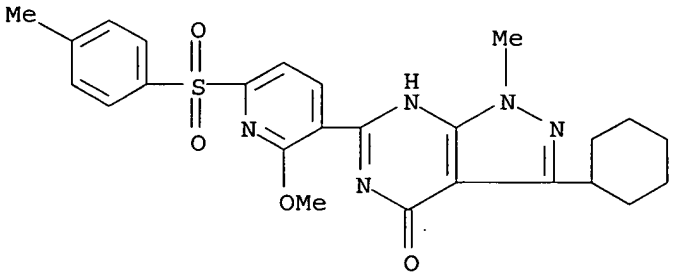
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-6-[2-methoxy-6-[(4-methylphenyl)sulfonyl]-3-pyridinyl]-1-methyl- (9CI) (CA INDEX NAME)



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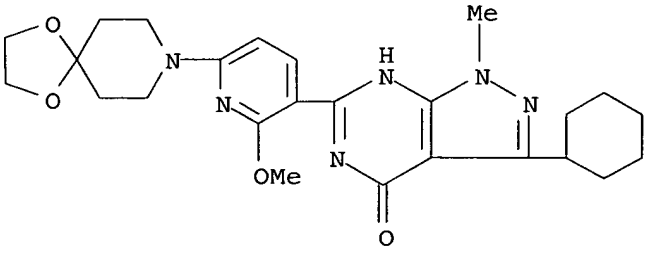

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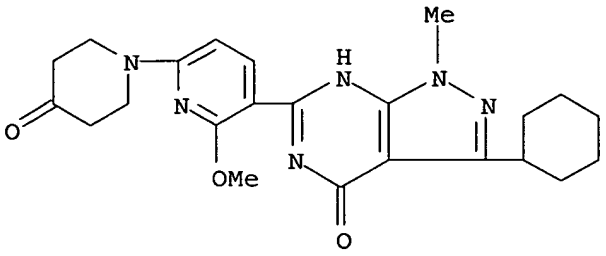

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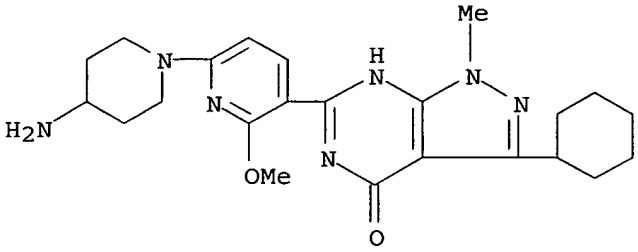

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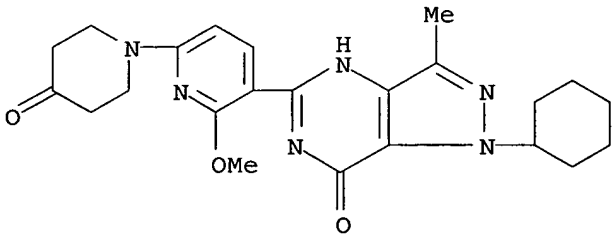
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[2-methoxy-6-(4-oxo-1-piperidinyl)-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



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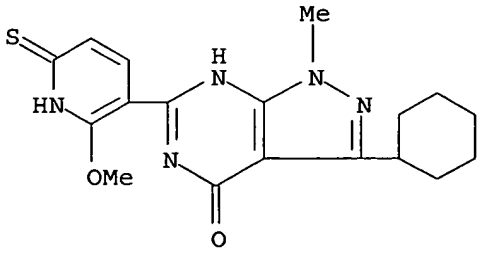

RN 812650-40-1 HCAPLUS
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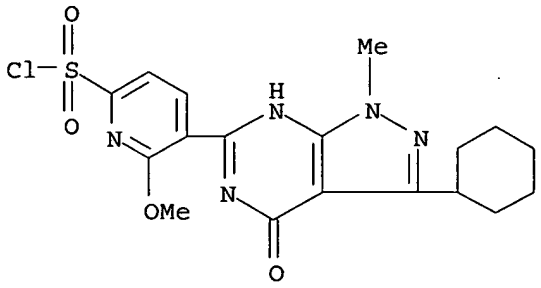

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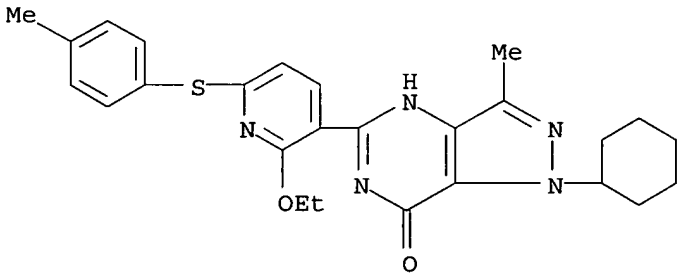

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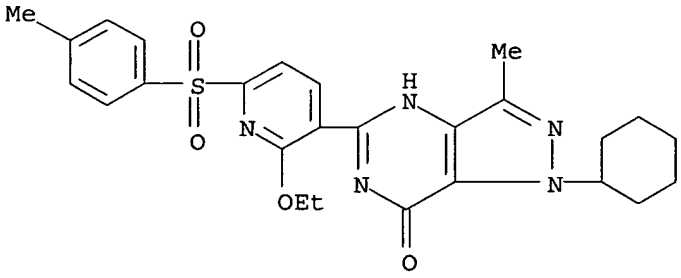

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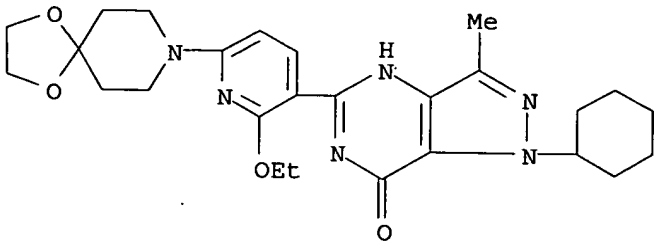

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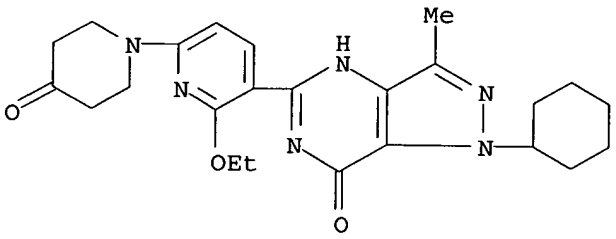

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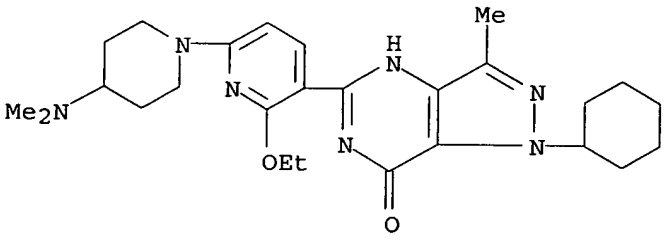

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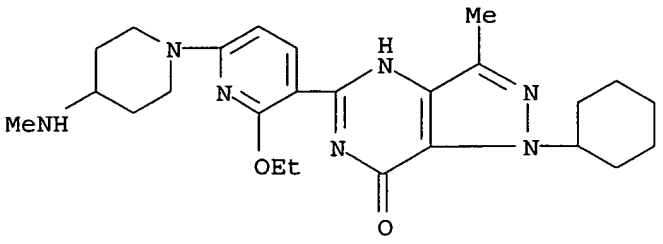

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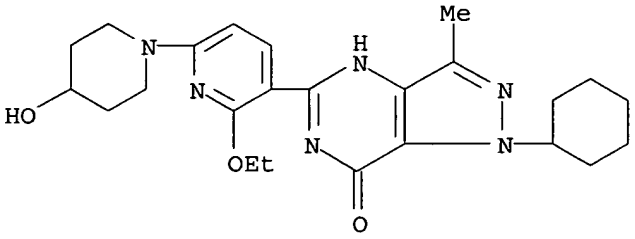

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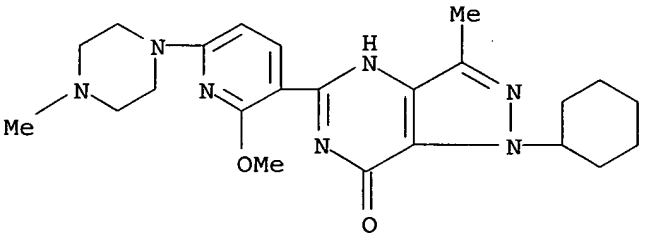
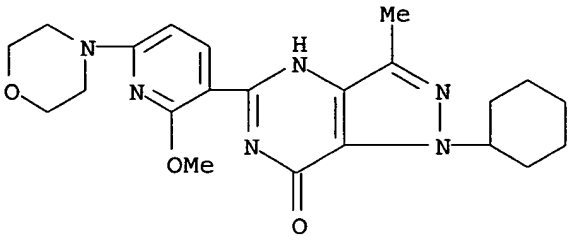
812650-27-4P 812650-30-9P 812650-31-0P
812650-32-1P 812650-34-3P 812650-35-4P
812650-37-6P 812650-38-7P 812650-41-2P
812650-42-3P 812650-43-4P 812650-48-9P
812650-49-0P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of pyridinyl pyrazolo[3,4-d]pyrimidin-4-ones and pyrazolo[4,3-d]pyrimidin-7-ones as PDE 7 inhibitors)

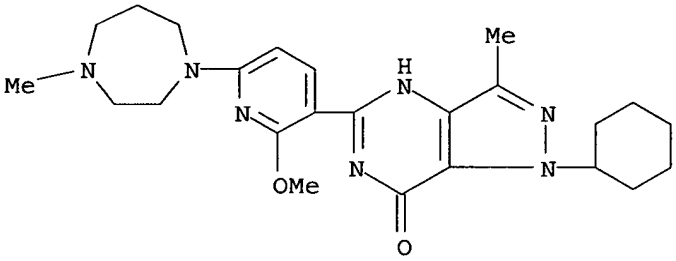
RN 812650-21-8 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[2-methoxy-6-(4-methyl-1-piperazinyl)-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



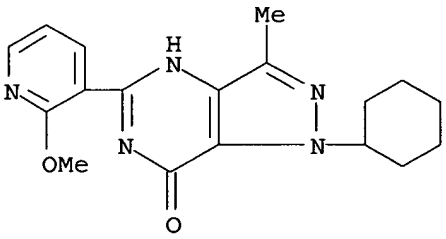
RN 812650-22-9 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[2-methoxy-6-(4-morpholinyl)-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



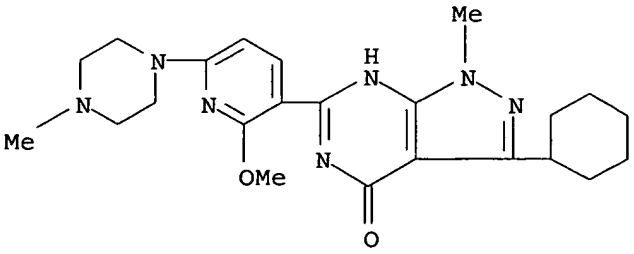
RN 812650-23-0 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-5-[6-(hexahydro-4-methyl-1H-1,4-diazepin-1-yl)-2-methoxy-3-pyridinyl]-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)



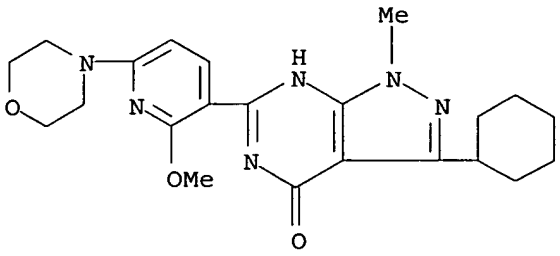
RN 812650-24-1 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-(2-methoxy-3-pyridinyl)-3-methyl- (9CI) (CA INDEX NAME)



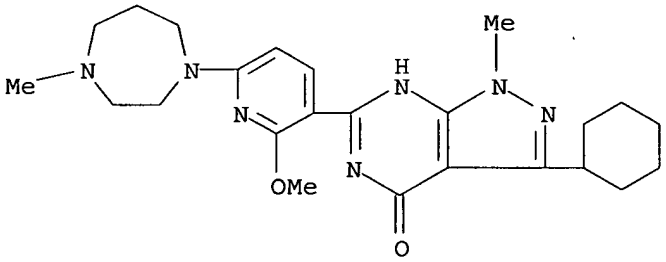
RN 812650-25-2 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-6-[2-methoxy-6-(4-methyl-1-piperazinyl)-3-pyridinyl]-1-methyl- (9CI) (CA INDEX NAME)



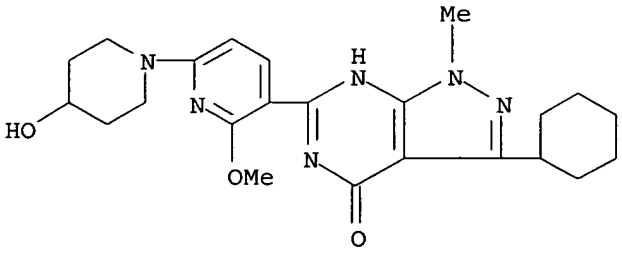
RN 812650-26-3 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-6-[2-methoxy-6-(4-morpholinyl)-3-pyridinyl]-1-methyl- (9CI) (CA INDEX NAME)



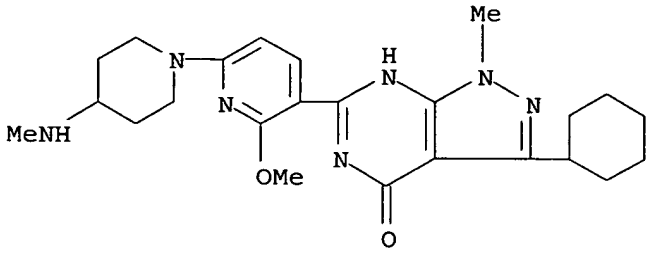
RN 812650-27-4 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-6-[6-(hexahydro-4-methyl-1H-1,4-diazepin-1-yl)-2-methoxy-3-pyridinyl]-1,5-dihydro-1-methyl- (9CI) (CA INDEX NAME)



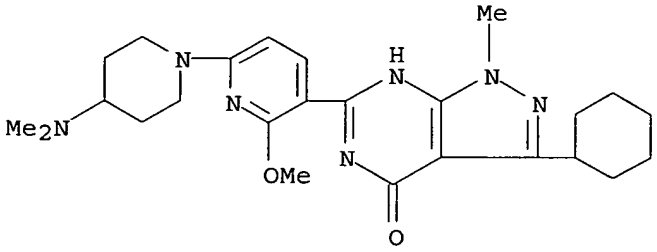
RN 812650-30-9 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-6-[6-(4-hydroxy-1-piperidinyl)-2-methoxy-3-pyridinyl]-1-methyl- (9CI) (CA INDEX NAME)



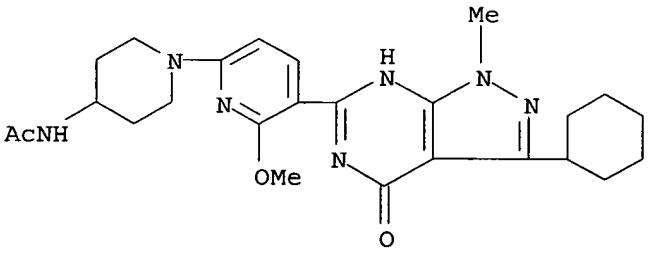
RN 812650-31-0 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-6-[2-methoxy-6-[4-(methylamino)-1-piperidinyl]-3-pyridinyl]-1-methyl- (9CI) (CA INDEX NAME)



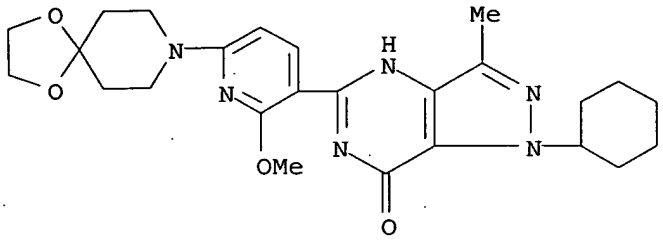
RN 812650-32-1 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-6-[6-[4-(dimethylamino)-1-piperidinyl]-2-methoxy-3-pyridinyl]-1,5-dihydro-1-methyl- (9CI) (CA INDEX NAME)



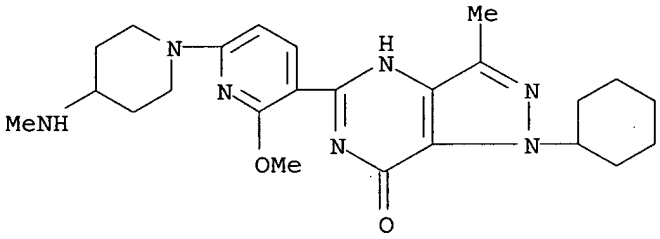
RN 812650-34-3 HCAPLUS
CN Acetamide, N-[1-[5-(3-cyclohexyl-4,5-dihydro-1-methyl-4-oxo-1H-pyrazolo[3,4-d]pyrimidin-6-yl)-6-methoxy-2-pyridinyl]-4-piperidinyl]- (9CI) (CA INDEX NAME)



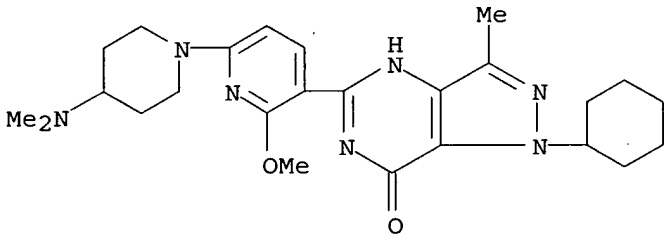
RN 812650-35-4 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-5-[6-(1,4-dioxa-8-azaspiro[4.5]dec-8-yl)-2-methoxy-3-pyridinyl]-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)



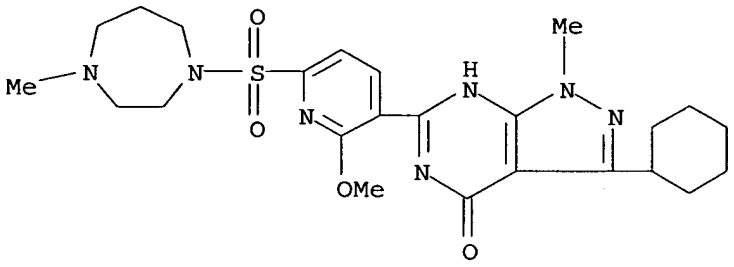
RN 812650-37-6 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[2-methoxy-6-[4-(methylamino)-1-piperidinyl]-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



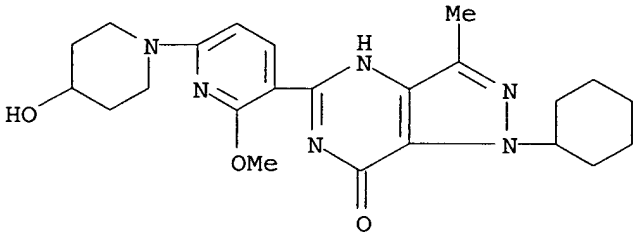
RN 812650-38-7 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-5-[6-[4-(dimethylamino)-1-piperidinyl]-2-methoxy-3-pyridinyl]-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)



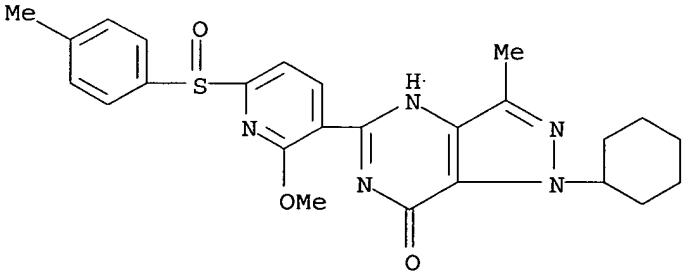
RN 812650-41-2 HCAPLUS
CN 1H-1,4-Diazepine, 1-[[5-(3-cyclohexyl-4,5-dihydro-1-methyl-4-oxo-1H-pyrazolo[3,4-d]pyrimidin-6-yl)-6-methoxy-2-pyridinyl]sulfonyl]hexahydro-4-methyl- (9CI) (CA INDEX NAME)



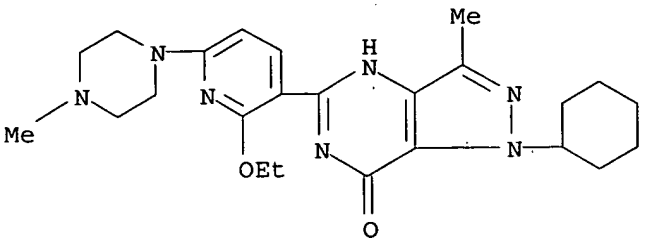
RN 812650-42-3 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[6-(4-hydroxy-1-piperidinyl)-2-methoxy-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



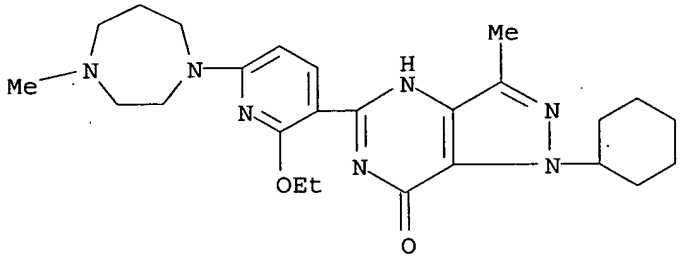
RN 812650-43-4 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-5-[2-methoxy-6-[(4-methylphenyl)sulfinyl]-3-pyridinyl]-3-methyl- (9CI) (CA INDEX NAME)



RN 812650-48-9 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-5-[2-ethoxy-6-(4-methyl-1-piperazinyl)-3-pyridinyl]-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)



RN 812650-49-0 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-5-[2-ethoxy-6-(hexahydro-4-methyl-1H-1,4-diazepin-1-yl)-3-pyridinyl]-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:511337 HCAPLUS
DOCUMENT NUMBER: 139:85373
TITLE: Preparation of pyrazolopyrimidinone derivatives having phosphodiesterase 7 (PDE7)-inhibitory activity
INVENTOR(S): Inoue, Hidekazu; Murafuji, Hidenobu; Hayashi, Yasuhiro
PATENT ASSIGNEE(S): Daiichi Suntory Pharma Co., Ltd., Japan; Suntory Limited; Daiichi Suntory Biomedical Research Ltd.
SOURCE: PCT Int. Appl., 244 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

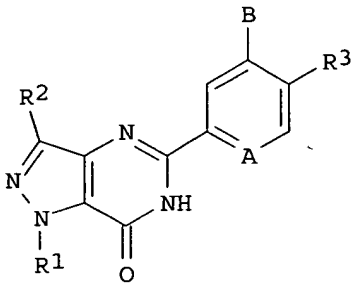
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003053975	A1	20030703	WO 2002-JP13083	20021213
W: BR, CA, CN, HU, JP, KR, US				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR				
CA 2439784	AA	20030703	CA 2002-2439784	20021213
BR 2002007215	A	20040210	BR 2002-7215	20021213
EP 1454897	A1	20040908	EP 2002-788833	20021213
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, CY, TR, BG, CZ, EE, SK				
CN 1533392	A	20040929	CN 2002-809154	20021213
US 2005148604	A1	20050707	US 2004-866198	20040614

Thom

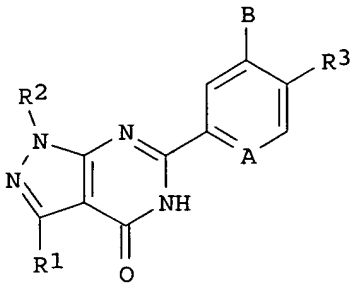
PRIORITY APPLN. INFO.: JP 2001-380483 A 20011213
WO 2002-JP13083 W 20021213

OTHER SOURCE(S): MARPAT 139:85373

GI



I



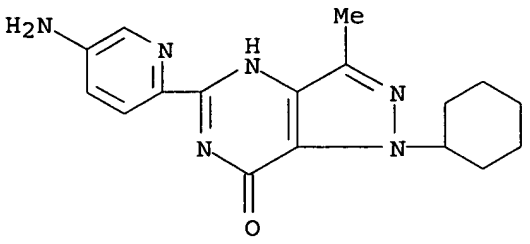
II

AB Pyrazolopyrimidinone derivs. represented by the general formula (I) or (II) [wherein A = N, CR4; wherein R4 = H, C1-3 alkoxy optionally substituted by ≥1 F atoms if necessary; B = H, halo; R1 = (un)substituted C3-7 cycloalkyl, tert-butyl; R2 = H, Me, Et; R3 = H, NO2, cyano, halo, NR5R6, C(:X)R7, SO2NR5R6, OR8, NR8CONR5R6, NR8SO2R9, heteroaryl, (un)substituted C1-3 alkyl; wherein R5, R6 = H, each (un)substituted C1-6 alkyl or acyl; or NR5R6 = azetidinyl, pyrrolidinyl, piperidinyl, morpholino, thiomorpholino, piperazinyl, or homopiperazinyl, each optionally substituted by (un)substituted C1-4 alkyl, OH, C1-3 alkoxy, CO2H, or NR5R6; R7 = (un)substituted C1-6 alkyl, OH, OR8, NR5R6; R8 = H, (un)substituted C1-6 alkyl; R9 = (un)substituted C1-6 alkyl; X = O, S, NH] or salts or solvates thereof are prepared These compds. have .apprx.10-times more potent activity for inhibiting PDE7 than PDE4, can enhance the intracellular cAMP level by virtue of their selective inhibitory activity against PDE7, and are useful in the prevention and treatment of various allergic diseases and inflammatory and immunol. diseases through their inhibiting the activation of T cells. Thus, 207 μL N-methylpiperazine, 120 mg sodium tert-butoxide, 12.6 mg tri(tert-butylphosphine), and 7.0 mg Pd(OAc)2 were added to a solution of 260 mg 6-(4-bromo-2-methoxyphenyl)-3-cyclohexyl-1-methyl-1,5-dihydro-4H-pyrazolo[3,4-d]pyrimidin-4-one in 8 mL toluene and refluxed for 5 h to give 85% 3-cyclohexyl-6-[2-methoxy-4-(4-methyl-1-piperazinyl)phenyl]-1-methyl-1,5-dihydro-4H-pyrazolo[3,4-d]pyrimidin-4-one (II). II.

IT **553668-79-4P**
RL: PAC (Pharmacological activity); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(preparation of pyrazolopyrimidinone derivs. as phosphodiesterase 7 (PDE7) inhibitors for prevention and treatment of various allergic diseases and inflammatory and immunol. diseases)

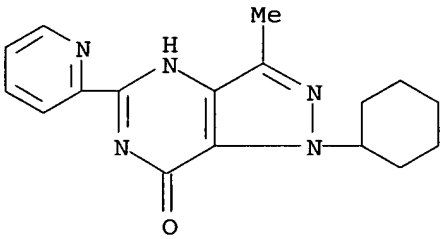
RN 553668-79-4 HCAPLUS

CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 5-(5-amino-2-pyridinyl)-1-cyclohexyl-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)

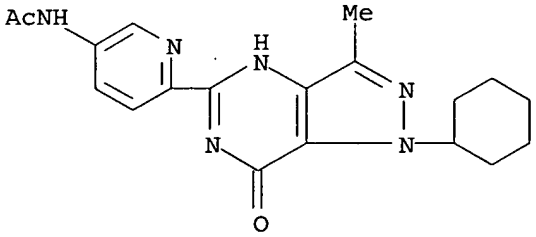


IT **553668-74-9P 553668-80-7P 553668-85-2P**
553668-93-2P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of pyrazolopyrimidinone derivs. as phosphodiesterase 7 (PDE7) inhibitors for prevention and treatment of various allergic diseases and inflammatory and immunol. diseases)

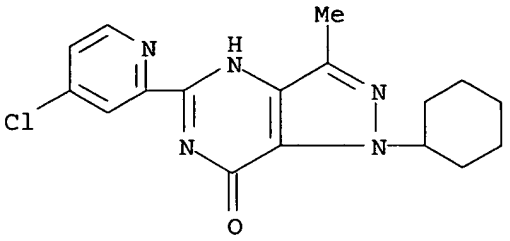
RN 553668-74-9 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-3-methyl-5-(2-pyridinyl)- (9CI) (CA INDEX NAME)



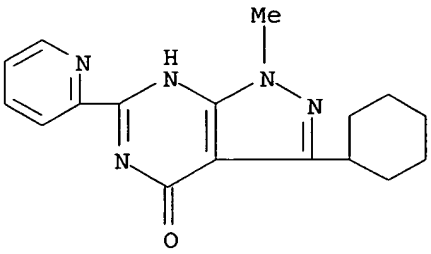
RN 553668-80-7 HCAPLUS
CN Acetamide, N-[6-(1-cyclohexyl-4,7-dihydro-3-methyl-7-oxo-1H-pyrazolo[4,3-d]pyrimidin-5-yl)-3-pyridinyl]- (9CI) (CA INDEX NAME)



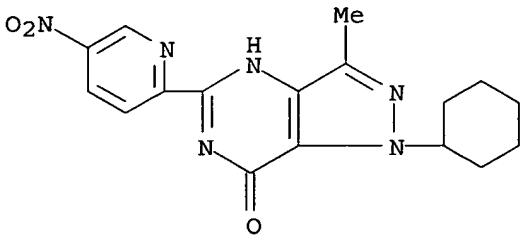
RN 553668-85-2 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 5-(4-chloro-2-pyridinyl)-1-cyclohexyl-1,4-dihydro-3-methyl- (9CI) (CA INDEX NAME)



RN 553668-93-2 HCAPLUS
CN 4H-Pyrazolo[3,4-d]pyrimidin-4-one, 3-cyclohexyl-1,5-dihydro-1-methyl-6-(2-pyridinyl)- (9CI) (CA INDEX NAME)



IT **553672-35-8P**, 1-Cyclohexyl-3-methyl-5-(5-nitro-2-pyridinyl)-1,6-dihydro-7H-pyrazolo[4,3-d]pyrimidin-7-one
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of pyrazolopyrimidinone derivs. as phosphodiesterase 7 (PDE7) inhibitors for prevention and treatment of various allergic diseases and inflammatory and immunol. diseases)
RN 553672-35-8 HCAPLUS
CN 7H-Pyrazolo[4,3-d]pyrimidin-7-one, 1-cyclohexyl-1,4-dihydro-3-methyl-5-(5-nitro-2-pyridinyl)- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:314395 HCAPLUS
DOCUMENT NUMBER: 136:335540
TITLE: Use of PDE V inhibitors for improved fecundity in mammals
INVENTOR(S): Westbrook, Simon Lempriere; Zanzinger, Johannes

Friedrich
PATENT ASSIGNEE(S): Pfizer Limited, UK; Pfizer Inc.
SOURCE: Eur. Pat. Appl., 20 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1199070	A2	20020424	EP 2001-308684	20011011
EP 1199070	A3	20040317		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
CA 2359383	AA	20020420	CA 2001-2359383	20011018
US 2003018036	A1	20030123	US 2001-982445	20011018
✓US 6548508	B2	20030415		
AU 2001081523	A5	20020502	AU 2001-81523	20011019
JP 2002220346	A2	20020809	JP 2001-322195	20011019
ZA 2001008617	A	20030422	ZA 2001-8617	20011019
NZ 514947	A	20050324	NZ 2001-514947	20011019
US 2003018037	A1	20030123	US 2002-229534	20020827
✓US 6743799	B2	20040601		
✓US 2004167095	A1	20040826	US 2004-778866	20040212
AU 2004233509	A1	20041223	AU 2004-233509	20041126

PRIORITY APPLN. INFO.:

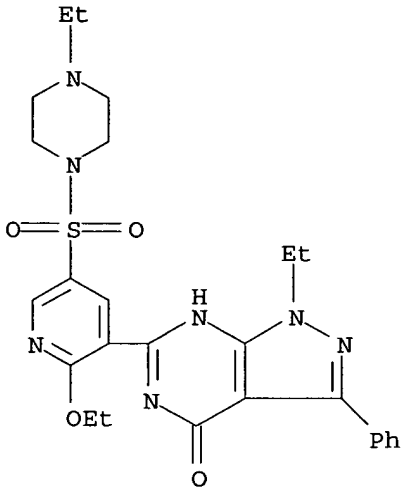
GB 2000-25782	A	20001020
US 2000-253338P	P	20001128
US 2001-982445	A1	20011018
AU 2001-81523	A3	20011019
US 2002-229534	A1	20020827

AB The invention relates to the use of a cyclic guanosine 3',5'-monophosphate phosphodiesterase type five (cGMP PDE V) inhibitor for increasing fecundity in a mammal by one or more of (a) promoting the growth of an oocyte, zygote, blastocyst, embryo and/or fetus, (b) increasing the rate or probability of survival of an embryo and/or fetus and (c) increasing the birth weight of a progeny, or for increasing milk productivity. I.v. and tablet formulations are exemplified. Formulations and packs containing the PDE V inhibitors for pharmaceutical or veterinary use are claimed.

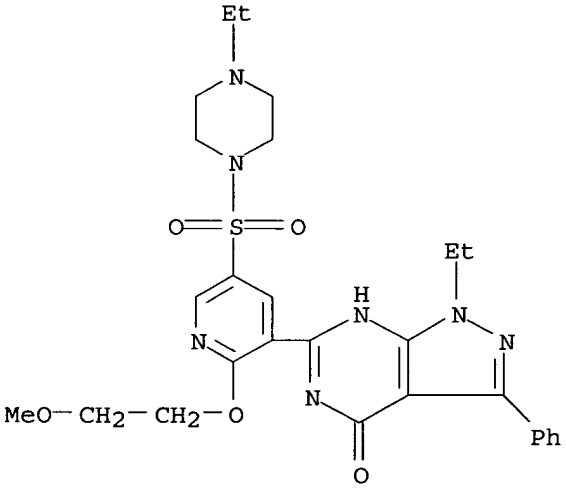
IT **264919-78-0 264919-79-1**
RL: AGR (Agricultural use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of PDE V inhibitors for improved fecundity in mammals)

RN 264919-78-0 HCAPLUS

CN Piperazine, 1-[[6-ethoxy-5-(1-ethyl-4,5-dihydro-4-oxo-3-phenyl-1H-pyrazolo[3,4-d]pyrimidin-6-yl)-3-pyridinyl]sulfonyl]-4-ethyl- (9CI) (CA INDEX NAME)



RN 264919-79-1 HCAPLUS
CN Piperazine, 1-ethyl-4-[[5-(1-ethyl-4,5-dihydro-4-oxo-3-phenyl-1H-pyrazolo[3,4-d]pyrimidin-6-yl)-6-(2-methoxyethoxy)-3-pyridinyl]sulfonyl]- (9CI) (CA INDEX NAME)

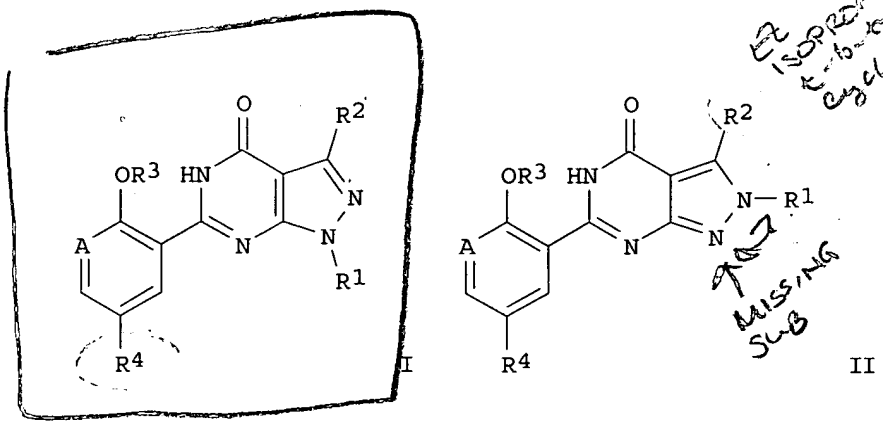


L18 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:277701 HCAPLUS
DOCUMENT NUMBER: 132:293775
TITLE: Preparation of pyrazolopyrimidinones as cGMP PDE5 inhibitors for the treatment of sexual dysfunction
INVENTOR(S): Bunnage, Mark Edward; Street, Stephen Derek Albert; Mathias, John Paul; Wood, Anthony
PATENT ASSIGNEE(S): Pfizer Inc., USA; Pfizer Limited
SOURCE: Eur. Pat. Appl., 40 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 995751	A2	20000426	EP 1999-308158	19991015
EP 995751	A3	20001018		
EP 995751	B1	20050629		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
AT 298753	E	20050715	AT 1999-308158	19991015
ES 2243037	T3	20051116	ES 1999-308158	19991015
CA 2287562	AA	20000423	CA 1999-2287562	19991022
BR 9905109	A	20000926	BR 1999-5109	19991022
US 6407114	B1	20020618	US 1999-425095	19991022
JP 2000128884	A2	20000509	JP 1999-302064	19991025
JP 3670908	B2	20050713		
MX 9909816	A	20000630	MX 1999-9816	19991025

PRIORITY APPLN. INFO.: GB 1998-23103 A 19981023
OTHER SOURCE(S): MARPAT 132:293775
GI

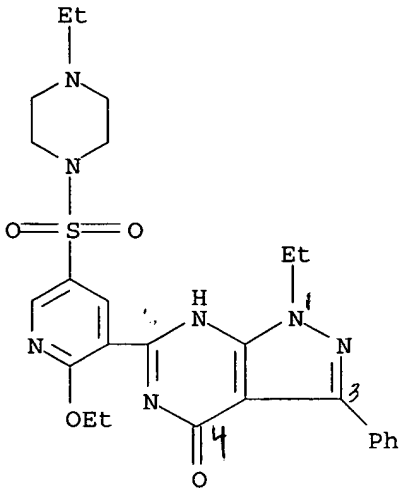


AB The title compds. [I or II; A = CH, N; R1, R2 = H, (un)substituted alkyl, (un)substituted Het, etc.; R3 = H, (un)substituted alkyl; R4 = SO2NR12R13; NR12R13 = Het; Het = 4-12 membered heterocyclic group containing at least one N atom and, optionally, one or more heteroatoms selected from N, S and O], useful in the curative and prophylactic treatment of a medical condition for which inhibition of a cyclic guanosine 3',5'-monophosphate phosphodiesterase (e.g. cGMP PDE5) is desired, were prepared E.g., a multi-step synthesis of I [A = CH; R1 = Pr; R2 = 2-pyridylmethyl; R3 = Pr; R4 = 4-ethylpiperazin-1-ylsulfonyl] which showed IC50 of 9.30 nM against cGMP PDE5, was given.

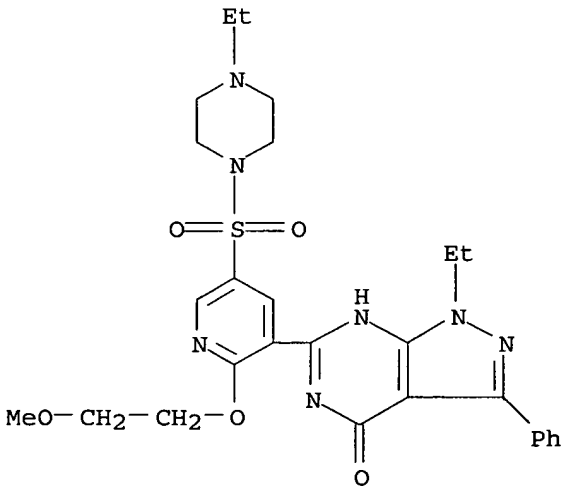
IT **264919-78-0P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(preparation of pyrazolopyrimidinones as cGMP PDE5 inhibitors for the treatment of sexual dysfunction)

RN 264919-78-0 HCAPLUS

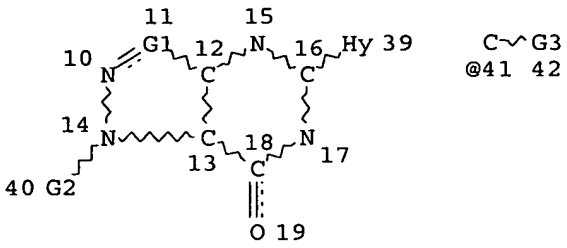
CN Piperazine, 1-[[6-ethoxy-5-(1-ethyl-4,5-dihydro-4-oxo-3-phenyl-1H-pyrazolo[3,4-d]pyrimidin-6-yl)-3-pyridinyl]sulfonyl]-4-ethyl- (9CI) (CA INDEX NAME)



IT **264919-79-1P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of pyrazolopyrimidinones as cGMP PDE5 inhibitors for the treatment of sexual dysfunction)
RN 264919-79-1 HCAPLUS
CN Piperazine, 1-ethyl-4-[[5-(1-ethyl-4,5-dihydro-4-oxo-3-phenyl-1H-pyrazolo[3,4-d]pyrimidin-6-yl)-6-(2-methoxyethoxy)-3-pyridinyl]sulfonyl]- (9CI) (CA INDEX NAME)



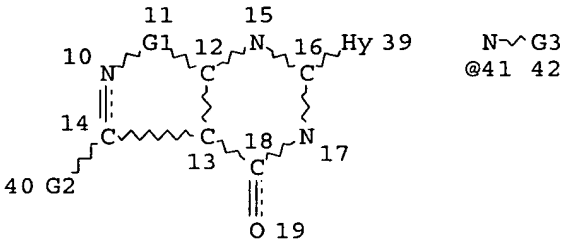
=> => d stat que l24
L12 STR



VAR G1=CH/41
VAR G2=CB/T-BU
VAR G3=ME/ET/I-PR/N-PR
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
GGCAT IS MCY AT 39
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 14

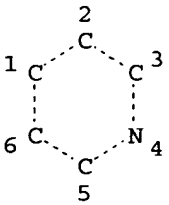
STEREO ATTRIBUTES: NONE
L13 STR



VAR G1=NH/41
VAR G2=CB/T-BU
VAR G3=ME/ET/I-PR/N-PR
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
GGCAT IS MCY AT 39
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 14

STEREO ATTRIBUTES: NONE
L15 46 SEA FILE=REGISTRY SSS FUL L12 OR L13
L16 STR



NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 6

STEREO ATTRIBUTES: NONE
L17 44 SEA FILE=REGISTRY SUB=L15 SSS FUL L16
L18 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L17
L19 47 SEA FILE=HCAPLUS ABB=ON PLU=ON "INOUE HIDEKAZU"/AU OR "INOUE HIDEKAZU C O DAINIPPON S"/AU
L21 14 SEA FILE=HCAPLUS ABB=ON PLU=ON ("MURAFUJI H"/AU OR "MURAFUJI HIDENOBU"/AU)
L22 1 SEA FILE=HCAPLUS ABB=ON PLU=ON "HAYASHI YASHIHIRO"/AU
L24 56 SEA FILE=HCAPLUS ABB=ON PLU=ON (L19 OR L21 OR L22) NOT L18

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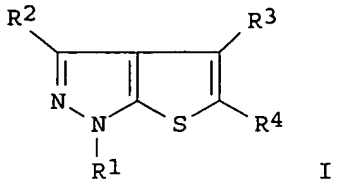
=> d ibib abs hitstr l24 1-56

L24 ANSWER 1 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:32180 HCAPLUS
DOCUMENT NUMBER: 144:128971
TITLE: Preparation of thienopyrazole derivatives as PDE7 inhibitors
INVENTOR(S): **Inoue, Hidekazu; Murafuji, Hidenobu** ; Hayashi, Yasuhiro
PATENT ASSIGNEE(S): Daiichi Asubio Pharma Co., Ltd., Japan
SOURCE: PCT Int. Appl., 329 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006004040	A1	20060112	WO 2005-JP12208	20050701

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: JP 2004-195836 A 20040701
GI



I

AB The title compds. I [R1 = (un)substituted alkyl, (un)substituted cycloalkyl, (un)substituted heterocycloalkyl; R2 = H, (un)substituted alkyl; R3 = H, (un)substituted alkyl, halo; R4 = (un)substituted aryl, (un)substituted heteroaryl, CO2R7, etc.; R7 = H, (un)substituted alkyl] are prepared I have selective inhibitory activity against PDE7 and thus heighten the intracellular cAMP level to inhibit the activation of T cells. I are hence useful in the prevention and treatment of various allergic diseases and inflammatory and immunol. diseases. Thus, N-benzyl-1-cyclohexyl-3-methyl-1H-thieno[2,3-c]pyrazole-5-carboxamide was prepared in a multistep process from cyclohexylhydrazine HCl salt and Me acetoacetate. Compds. of this invention showed IC50 values of 0.004 μM to 0.009 μM against phosphodiesterase 7.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 2 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1081858 HCAPLUS

DOCUMENT NUMBER: 144:105301

TITLE: Functional expression of a proliferation-related ligand in hepatocellular carcinoma and its implications for neovascularization

AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; Yamanaka, Yutaka; **Inoue, Hidekazu**; Kawakita, Tomoyuki; Saitou, Yukiko; Yamaguchi, Yumi; Enokimura, Naoyuki; Ito, Keiichi; Yamamoto, Norihiko; Sugimoto, Kazushi; Murata, Kazumoto; Nakano, Takeshi

CORPORATE SOURCE: Department of Internal Medicine, Mie University School of Medicine, Tsu, 514-8507, Japan

SOURCE: World Journal of Gastroenterology (2005), 11(30), 4650-4654
CODEN: WJGAF2; ISSN: 1007-9327

PUBLISHER: World Journal of Gastroenterology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB AIM: To detect the expression of a proliferation-related ligand on human hepatocellular carcinoma (HCC) cell lines (SK-Hep1, HLE and HepG2) and in culture medium. METHODS: APRIL expression was analyzed by Western blotting in HCC cell lines. Effects of APRIL to cell count and angiogenesis were analyzed, too. RESULTS: Recombinant human APRIL (rhAPRIL) increased cell viability of HepG2 cells and, in HUVEC, rhAPRIL provided slight tolerance to cell death from serum starvation. Soluble APRIL (sAPRIL) from HLE cells increased after serum starvation, but did not change in SK-Hep1 or HepG2 cells. These cells showed down-regulation of VEGF after incubation with anti-APRIL antibody. Furthermore, culture medium from the HCC cells treated with anti-APRIL antibody treatment inhibited tube formation of HUVECs. CONCLUSION: Functional expression of APRIL might contribute to neovascularization via an upregulation of VEGF in HCC.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 3 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:804651 HCAPLUS
TITLE: Substrate processing apparatus
INVENTOR(S): Sasada, Shigeru C. O. Dainippon S.; Aoki, Kaoru C. O. Dainippon Scree; Kodama, Mitsumasa C. O. Dainippon; Sugimoto, Kenji C. O. Dainippon S.; Fukumoto, Yoshiteru C. O. Dainipp; **Inoue, Hidekazu C. O. Dainippon S.**
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Eur. Pat. Appl., No pp. given
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 688041	A1	19951220	EP 1995-108522	19950602
R: DE, FR, GB, IT				
KR 171866	B1	19990330	KR 1995-16058	19950616

PRIORITY APPLN. INFO.: JP 1994-135796 A 19940617
AB Unavailable

L24 ANSWER 4 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:731539 HCAPLUS
DOCUMENT NUMBER: 143:174531
TITLE: Release films for fabrication of (flexible/multilayer) printed circuit boards and manufacture thereof
INVENTOR(S): Matsumoto, Hirotake; Shirado, Hitoshi; **Inoue, Hidekazu**
PATENT ASSIGNEE(S): Sekisui Chemical Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 14 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2005212453	A2	20050811	JP 2004-25824	20040202

PRIORITY APPLN. INFO.: JP 2004-25824 20040202
AB The films, having release layers satisfying blocking strength (ASTM D 1893; 170°, 3 MPa, 30 min) against the same layer ≤0.02 N/cm and comprised of polar group-containing matrix resins with halo content ≤5%, are manufactured by heat treatment of films from the resin compns. at Tg-Tm (Tg, Tm = glass transition temperature and m.p. of the matrix resins, resp.). Thus, Hytrel 2751 (halo-free resin composition; Tg 53°) was kneaded at 250°, extruded through a T die, and passed through a pair of hot rolls at 170° to give a 50-μm-thick release film showing storage modulus 130 MPa (170°) and 1800 MPa at 23°, resp., tensile breaking elongation 1400% at 170°, and blocking strength 0.015 N/cm.

L24 ANSWER 5 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:730763 HCAPLUS
DOCUMENT NUMBER: 143:339121

TITLE: Vitamin K analog (compound 5) induces apoptosis in human hepatocellular carcinoma independent of the caspase pathway

AUTHOR(S): Enokimura, Naoyuki; Shiraki, Katsuya; Kawakita, Tomoyuki; Saitou, Yukiko; **Inoue, Hidekazu**; Okano, Hiroshi; Yamamoto, Norihiko; Sugimoto, Kazushi; Carr, Brian I.; Nakano, Takeshi

CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Mie, Japan

SOURCE: Anti-Cancer Drugs (2005), 16(8), 837-844
CODEN: ANTDEV; ISSN: 0959-4973

PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A systemic vitamin K analog, compound 5 (Cpd 5), possesses the ability to inhibit cell growth of tumor cells. Therefore, we investigated the effect of Cpd 5 in human hepatocellular carcinoma (HCC) cell lines and evaluated its role in apoptosis. Human HCC cell lines were cultured and treated with Cpd 5. Apoptosis was assessed using DAPI staining and Annexin-V membrane staining. The expression of caspases, XIAP and Bcl-xL was also investigated. Cpd 5 decreased cell viability in a dose-dependent manner in two HCC cells (HLE and SK-Hep1) containing mutant p53, but not in the HepG2 cell line, which contained wild-type p53. Cpd 5-treated HLE and SK-Hep1 cells showed typical apoptotic features, nuclear condensation and nuclear fragmentation upon DAPI staining. Pos. membranous staining for Annexin-V was also seen in these cells. Both caspase-8 and caspase-3 activities were up-regulated slightly. Pro-caspase-8 protein levels decreased slightly in both cells. Although the expression of Bcl-xL was not influenced by Cpd 5, that of XIAP decreased in HLE cells. However, the pan-caspase inhibitor, zVAD, could not significantly prevent Cpd 5-induced apoptosis and Cpd 5 could not augment TRAIL-induced apoptosis. These results demonstrate that Cpd 5 induced apoptosis in human HCC cell lines, mainly independently of caspase activities. This may contribute to its highly potent cytotoxicity toward HCC cells.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 6 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:638924 HCAPLUS

DOCUMENT NUMBER: 143:134528

TITLE: Mold release film for printed circuit boards

INVENTOR(S): Matsumoto, Hirotake; Shirato, Hitoshi; **Inoue, Hidekazu**

PATENT ASSIGNEE(S): Sekisui Chemical Co., Ltd., Japan

SOURCE: PCT Int. Appl., 28 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005066246	A1	20050721	WO 2003-JP16905	20031226

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: WO 2003-JP16905 20031226

AB The mold release film with good flexibility, unevenness follow-up property, thermal stability, mold release capability, and non-staining property a has on ≥1 surface a layer derive from a resin composition containing a matrix resin having polar groups on its main chain and having halogen content ≤5 %. Stacking sequentially a release film of Hytrel 2751 (polyester rubber), a Cu foil-clad polyimide film (Kapton), a coverlay film of polyimide (Kapton), a release film, and an LDPE film (Novatec LE425), hot pressing, and removing the release film and the LDPE film gave a flexible printed circuit board.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 7 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:407810 HCAPLUS

DOCUMENT NUMBER: 143:130781

TITLE: Expression of TNF-related apoptosis-inducing ligand in human hepatocellular carcinoma

AUTHOR(S): Shiraki, Katsuya; Yamanaka, Takenari; **Inoue, Hidekazu**; Kawakita, Tomoyuki; Enokimura, Naoyuki; Okano, Hiroshi; Sugimoto, Kazushi; Murata, Kazumoto; Nakano, Takeshi

CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, Mie, 514-8507, Japan

SOURCE: International Journal of Oncology (2005), 26(5), 1273-1281
CODEN: IJONES; ISSN: 1019-6439

PUBLISHER: International Journal of Oncology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB TNF-related apoptosis-inducing ligand (TRAIL), as well as Fas ligand, plays a pivotal role in lymphocyte cytotoxicity and the maintenance of immunol. homeostasis in various tissues, but its physiol. role in immune evasion of cancer cells remains unknown. We have previously shown strong resistance to TRAIL-induced cytotoxicity in human hepatocellular carcinomas (HCCs). The current study investigates the expression of TRAIL in HCCs. We found that three HCC cells, HepG2, Hep3B and Huh7 cells, constitutively express TRAIL mRNA and protein, as detected by reverse transcriptase PCR and Western blotting. Four of 10 human HCC tissues demonstrated pos. staining for TRAIL, whereas non-tumor tissues showed little detectable staining. TRAIL expression on tumor cells was detected by flow cytometry and was dramatically induced after the addition of doxorubicin, a chemotherapeutic agent, or cytokine stimulation with TNF-α, IL-16 or IL-18. This expression was induced principally via the NF-κB activation pathway, since IκB transfection significantly reduced TRAIL expression. In addition, the expressed TRAIL was functional. The TRAIL on HCC cells induced apoptosis in Jurkat cells that are sensitive to TRAIL-mediated apoptosis, and this process was specifically inhibited by recombinant TRAIL-receptors:Fc which binds to TRAIL. In conclusion, TRAIL expressed on the surface of HCC cells by cytokines or cytostatic drugs might contribute to an alternative mechanism that enables tumors to evade immune surveillance by inducing apoptosis of activated human lymphocytes.

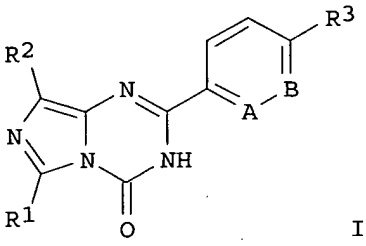
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 8 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:1127383 HCAPLUS
DOCUMENT NUMBER: 142:74617
TITLE: Imidazotriazinone derivatives as PDE 7 (phosphodiesterase 7) inhibitors, their preparation, and pharmaceutical compositions containing them
INVENTOR(S): **Inoue, Hidekazu; Murafuji, Hidenobu** ; Hayashi, Yasuharu
PATENT ASSIGNEE(S): Daiichi Suntory Pharma Co.,ltd., Japan; Daiichi Suntory Biomedical Research Co.,ltd.
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

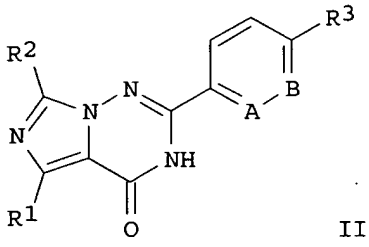
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004111053	A1	20041223	WO 2004-JP8642	20040611

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

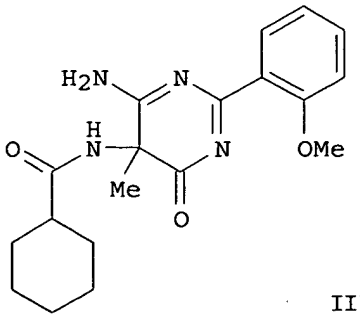
PRIORITY APPLN. INFO.: JP 2003-170095 A 20030613
OTHER SOURCE(S): MARPAT 142:74617
GI



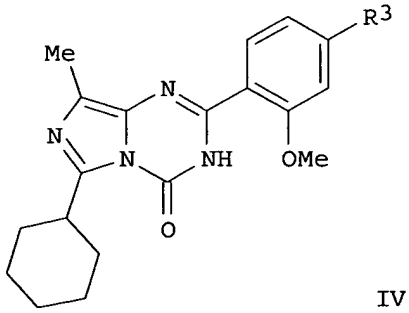
I



II



III



IV

AB The invention provides compds. which inhibit PDE 7 selectively, and therefore enhance cellular cAMP levels. Consequently, the compds. are useful for treating various kinds of diseases, such as allergic diseases, inflammatory diseases, or immunol. diseases. The compds. are imidazotriazinones I and II [wherein: A is N or CR4; B is N or CH; R1 is (un)substituted cycloalkyl or tert-Bu; R2 is H or C1-C6 alkyl; R3 is H, NO2, cyano, halo, heteroaryl, (un)substituted C1-C6 alkyl, (un)substituted C2-C6 alkenyl, (un)saturated (un)substituted heterocycloalkyl, NR5R6, COR7, SO2R7, OR8, NR8COR7, NR8SO2R7; R4 is H or C1-C3 alkoxy group which is (un)substituted by one or more F atom(s); R5 and R6 are (independently) H, (un)substituted C1-C6 alkyl, (un)substituted acyl, or (un)substituted heterocycloalkyl; R7 is H, (un)substituted C1-C6 alkyl group, (un)substituted heterocycloalkyl, OH, OR8, or NR5R6; R8 is H, (un)substituted C1-C6 alkyl, or (un)substituted heterocycloalkyl; or pharmaceutically acceptable salts or solvates]. The compds. include particularly I and II [wherein: R1 is cyclohexyl; R2 is Me; R3 is H, NO2, cyano, halo, heteroaryl, (un)substituted C1-6 alkyl, (un)substituted C2-6 alkenyl, (un)saturated heterocycloalkyl, NR5R6, COR7, SO2R7, OR8, NR8COR7, NR8SO2R7; A is CR4; and B is CH]. The prepared compds. include 4 invention compds. and 8 intermediates. For instance, amidation of Et aminocyanoacetate with cyclohexanecarbonyl chloride gave 71% Et cyano[(cyclohexylcarbonyl)amino]acetate, which was methylated using NaOEt and MeI to give 88% Et 2-cyano-2-[(cyclohexylcarbonyl)amino]propanoate. The latter compound was cyclocondensed with 2-methoxybenzamidine HCl to give 21% pyrimidinone intermediate III, which was cyclized by treatment with Me3SiCl and then HMDS to give invention compound IV [R3 = H]. The exptl. inhibition of human PDE 7 (IC50) was 0.34 μM for IV [R3 = H] and 0.055 μM for IV [R3 = 4-methylpiperazin-1-yl]. The invention compds. inhibited PDE 7 with a selectivity of more than 10 times compared to PDE 4.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 9 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:669335 HCAPLUS
DOCUMENT NUMBER: 141:364885
TITLE: Adenoviral-mediated transfer of p53 gene enhances TRAIL-induced apoptosis in human hepatocellular carcinoma cells
AUTHOR(S): **Inoue, Hidekazu**; Shiraki, Katsuya; Murata, Kazumoto; Sugimoto, Kazushi; Kawakita, Tomoyuki; Yamaguchi, Yumi; Saitou, Yukiko; Enokimura, Naoyuki; Yamamoto, Norihiko; Yamanaka, Yutaka; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Mie, 514-8507, Japan
SOURCE: International Journal of Molecular Medicine (2004), 14(2), 271-275
CODEN: IJMMFG; ISSN: 1107-3756
PUBLISHER: International Journal of Molecular Medicine
DOCUMENT TYPE: Journal
LANGUAGE: English

AB P53 is a tumor suppressor protein with numerous biol. functions including transformation, regulation of cell growth, differentiation and apoptosis. The TNF-related apoptosis-inducing ligand (TRAIL) can induce apoptosis in various transformed cell lines. The authors investigated the effects of combining wild-type p53 gene transduction by adenoviral infection (Ad-p53) with addition of TRAIL on cell death, expression levels of TRAIL receptors (TRAIL-R1, TRAIL-R2), FLICE inhibitory protein (FLIP) and X-linked

inhibitor of apoptosis protein (XIAP) on human hepatocellular carcinoma (HCC) cell lines. HCC cell death was increased by combination of Ad-p53 infection and addition of TRAIL compared to either alone. Western blotting demonstrated decreased TRAIL-R1 and TRAIL-R2 levels after infection with Ad-p53. FLIP levels decreased in Huh7 cells and Hep3B cells, and XIAP levels decreased in all three HCC cell lines after infection with Ad-p53. Thus, death of HCC cells due to combined p53 gene transduction and exogenous TRAIL may be due to down regulation of FLIP or XIAP.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 10 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:992435 HCAPLUS
DOCUMENT NUMBER: 140:211277
TITLE: The PPARγ ligand, 15-deoxy-Δ12,14-PGJ2, regulates apoptosis-related protein expression in cholangio cell carcinoma cells
AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; **Inoue, Hidekazu**; Kawakita, Tomoyuki; Deguchi, Masatoshi; Sugimoto, Kazushi; Sakai, Takahisa; Murata, Kazumoto; Nakano, Takeshi; Enjoji, Munechika
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, 514-8507, Japan
SOURCE: International Journal of Molecular Medicine (2003), 12(6), 867-870
CODEN: IJMMFG; ISSN: 1107-3756
PUBLISHER: International Journal of Molecular Medicine
DOCUMENT TYPE: Journal
LANGUAGE: English

AB PPARγ is known to induce apoptosis in malignant tumor cells, but the mechanism of this induction is not well understood. We investigated induction of apoptosis with 15-deoxy-Δ12,14-prostaglandin J2 (15d-PGJ2), a PPARγ ligand, in cholangio cell carcinoma (CCC) cells (RBE, ETK-1 or HuCCT-1). Apoptosis was induced in RBE and ETK-1 cells with 15d-PGJ2, but not in HuCCT-1 cells, although PPARγ was expressed in all CCC cells. Apoptosis-related proteins were also expressed, including FLIP, bclx, Apaf-1 and XIAP, but expression levels differed among the three cell lines. RBE cells treated with 15d-PGJ2 showed caspase activation, and it appeared that PPARγ-induced apoptosis was dependent on caspase activation. However, neither ETK-1 nor HuCCT-1 cells showed significant activation of caspase-8 or -3 with 15d-PGJ2 treatment, raising the possibility of a caspase-independent apoptosis induction pathway. XIAP was down-regulated by 15d-PGJ2 in all three CCC cell lines. Therefore, 15d-PGJ2 induces apoptosis in CCC cells via caspase-dependent or independent pathways. 15D-PGJ2 may also induce down-regulation of XIAP and may promote caspase cascade activation through TNF-family receptor signaling pathways.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 11 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:782073 HCAPLUS
DOCUMENT NUMBER: 140:263902
TITLE: 15-Deoxy-Δ-12-14-PGJ2 Regulates Apoptosis Induction and Nuclear Factor-κB Activation Via a Peroxisome Proliferator-Activated Receptor-γ-Independent Mechanism in Hepatocellular Carcinoma
AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; **Inoue, Hidekazu**; Yamanaka, Yutaka; Kawakita, Tomoyuki; Saitou, Yukiko; Yamaguchi, Yumi; Enokimura, Naoyuki;

Yamamoto, Norihiko; Sugimoto, Kazushi; Murata, Kazumoto; Nakano, Takeshi
CORPORATE SOURCE: Dept. of Internal Medicine, Mie Univ. School of Medicine, Tsu, Japan
SOURCE: Laboratory Investigation (2003), 83(10), 1529-1539
CODEN: LAINAW; ISSN: 0023-6837
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The peroxisome proliferator-activated receptor-γ (PPARγ) high-affinity ligand, 15-deoxy-Δ-12,14-PGJ2 (15d-PGJ2), is toxic to malignant cells through cell cycle arrest and apoptosis induction. In this study, we investigated the effects of 15d-PGJ2 on apoptosis induction and expression of apoptosis-related proteins in hepatocellular carcinoma (HCC) cells. 15d-PGJ2 induced apoptosis in SK-Hep1 and HepG2 cells at a 50 μm concentration Pretreatment with the pan-caspase inhibitor, benzyloxycarbonyl-Val-Ala-Asp (OMe) fluoromethyl ketone (2-VAD-fmk), only partially blocked apoptosis induced by 40 μm 15d-PGJ2. This indicated that 15d-PGJ2 induction of apoptosis was associated with a caspase-3-independent pathway. 15d-PGJ2 also induced down-regulation of the X chromosome-linked inhibitor of apoptosis (XIAP), Bclx, and apoptotic protease-activating factor-1 in SK-Hep1 cells but not in HepG2 cells. However, 15d-PGJ2 sensitized both HCC cell lines to TNF-related apoptosis-induced ligand-induced apoptosis. In SK-Hep1 cells, cell toxicity, nuclear factor-κB (NF-κB) suppression, and XIAP down-regulation were induced by 15d-PGJ2 treatment under conditions in which PPARγ was down-regulated. These results suggest that the effect of 15d-PGJ2 was through a PPARγ-independent mechanism. Although cell toxicity was induced when PPARγ was down-regulated in HepG2 cells, NF-κB suppression and XIAP down-regulation were not induced. In conclusion, 15d-PGJ2 induces apoptosis of HCC cell lines via caspase-dependent and -independent pathways. In SK-Hep1 cells, the ability of 15d-PGJ2 to induce cell toxicity, NF-κB suppression, or XIAP down-regulation seemed to occur via a PPARγ-independent mechanism, but in HepG2 cells, NF-κB suppression by 15d-PGJ2 was dependent on PPARγ.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 12 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:691376 HCAPLUS
DOCUMENT NUMBER: 139:336097
TITLE: Fas stimulation activates NF-κB in SK-Hep1 hepatocellular carcinoma cells
AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; **Inoue, Hidekazu**; Kawakita, Tomoyuki; Saitou, Yukiko; Enokimura, Naoyuki; Yamamoto, Norihiko; Sugimoto, Kazushi; Murata, Kazumoto; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Mie, 514-8507, Japan
SOURCE: Oncology Reports (2003), 10(5), 1145-1148
CODEN: OCRPEW; ISSN: 1021-335X
PUBLISHER: Oncology Reports
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The TNF-receptor family has a dual signaling pathway, including induction of apoptosis and NF-κB activation associated with cell survival. Hepatocellular carcinoma (HCC) cells express TNF-receptor family members and the signaling from these receptors induces NF-κB activation. However, the role of Fas in induction of NF-κB activation in HCC

cells is not well understood. In this study, SK-Hep1, HepG2 or HLE cells were stimulated by anti-Fas agonistic antibody. Fas stimulation induced NF-κB activation in a dose-dependent manner in SK-Hep1 and HepG2 cell lines, but not in HLE cells. Anti-Fas agonistic antibody or the metabolic inhibitor, cyclo-heximide (CHX), failed to kill SK-Hep1 cells, but co-incubation with anti-Fas agonistic antibody and CHX was effective for induction of apoptosis. SK-Hep1 cell lines receiving Fas stimulation had increased viability, but the extent of cell proliferation was not dose-dependent. The observation suggests that Fas stimulation may contribute to HCC cell survival or proliferation.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 13 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:576153 HCAPLUS
DOCUMENT NUMBER: 139:185858
TITLE: Photocatalytic dehalogenation coupled on-Line to a reversed micellar-mediated chemiluminescence detection system: application to the determination of iodinated aromatic compounds
AUTHOR(S): Fujiwara, Terufumi; Mohammadzai, Imdad U.; **Inoue, Hidekazu**; Shimizu, Yasuhide; Kumamaru, Takahiro
CORPORATE SOURCE: Department of Chemistry, Graduate School of Science, Hiroshima University, Higashi-Hiroshima, 739-8526, Japan
SOURCE: Analytical Chemistry (2003), 75(17), 4493-4498
CODEN: ANCHAM; ISSN: 0003-2700
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of illumination time, temperature, catalyst concentration, and pH on the online photocatalytic dehalogenation of iodinated aromatic compds. in a near-UV-illuminated titanium dioxide (anatase type) aqueous suspension were monitored via the iodine-luminol chemiluminescence (CL) reaction in a reversed micellar medium, and a new, automated, rapid, and efficient method was developed. A water-cooled, 400-W high-pressure Hg lamp was used as an internal light source. The flow procedure involved the following: (1) photocatalytic dehalogenation/degradation of the iodinated compound by the near-UV-illuminated titanium dioxide and the production of iodide species, (2) oxidation of iodide into iodine, (3) extraction of iodine into cyclohexane, (4) membrane separation of the iodine-containing organic phase from the aqueous phase, and (5) the detection of iodine using the luminol CL reaction in the reversed micellar solution of cetyltrimethylammonium chloride in 6:5 (volume/volume) chloroform-cyclohexane/water buffered with sodium carbonate. Results for the dehalogenation of the iodinated compds., o-iodobenzoic acid and L-thyroxine (3,5,3',5'-tetraiodothyronine) sodium, were compared with a standard inorg. iodide solution After establishing the optimum chemical and instrumental conditions, detection limits of 0.8 + 10-9 and 0.2 + 10-9 M and linear calibration graphs were obtained with dynamic ranges from 0.79 + 10-7 to 7.9 + 10-7 M and from 0.20 + 10-7 to 2.0 + 10-7 M for o-iodobenzoic acid and L-thyroxine, resp. A precision of .apprx.4% relative standard deviation (n = 6) was provided at an o-iodobenzoic acid concentration of 0.79 + 10-7 M. The method developed was applied to the online detns. of iodinated aromatic compds. such as L-thyroxine sodium and iopamidol ((S)-N,N'-bis[2-hydroxy-1-(hydroxymethyl)ethyl]-5-[(2-hydroxy-1-oxopropyl)amino]-2,4,6-

triiodoisophthaldiamide) in pharmaceuticals.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 14 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:521303 HCAPLUS
DOCUMENT NUMBER: 139:274698
TITLE: Cellular FLICE/Caspase-8-Inhibitory Protein as a Principal Regulator of Cell Death and Survival in Human Hepatocellular Carcinoma
AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; **Inoue, Hidekazu**; Kawakita, Tomoyuki; Yamanaka, Takenari; Deguchi, Masatoshi; Sugimoto, Kazushi; Sakai, Takahisa; Ohmori, Shigeru; Fujikawa, Katsuhiko; Murata, Kazumoto; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Mie, Japan
SOURCE: Laboratory Investigation (2003), 83(7), 1033-1043
CODEN: LAINAW; ISSN: 0023-6837
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human hepatocellular carcinomas (HCCs) show resistance to apoptosis mediated by several death receptors. Because cellular FLICE/caspase-8-inhibitory protein (cFLIP) is a recently identified intracellular inhibitor of caspase-8 activation that potently inhibits death signaling mediated by all known death receptors, including Fas, TNF-receptor (TNF-R), and TNF-related apoptosis-inducing ligand receptors (TRAIL-Rs), we investigated the expression and function of cFLIP in human HCCs. We found that cFLIP is constitutively expressed in all human HCC cell lines and is expressed more in human HCC tissues than in nontumor liver tissues. Metabolic inhibitors, actinomycin D (ActD) or cycloheximide (CHX), dramatically rendered HCC cells sensitive to Fas-mediated apoptosis. Neither caspase-8 nor caspase-3 was activated by agonistic anti-Fas antibody alone, but both caspases were activated by Fas stimulation in the presence of ActD or CHX, indicating the importance of caspase-8 inhibitors that are sensitive to metabolic inhibitors. Actually, cFLIP expression was decreased in ActD or CHX treatment. CFLIP down-regulation induced by cFLIP antisense oligodeoxynucleotides sensitized HLE cells to Fas, TNF-R, and TRAIL-R-mediated apoptosis. Furthermore, cFLIP over-expression activated nuclear factor (NF)-κB and cFLIP down-regulation attenuated NF-κB activation induced by TNF-α or TRAIL. Pretreatment with pan-caspase-inhibitor, benzyloxycarbonyl-Val-Ala-Asp (OMe) fluoromethyl ketone (Z-VAD-fmk), restored NF-κB activity attenuated by cFLIP down-regulation. CFLIP expression was increased by TNF-, TRAIL, or vascular endothelial growth factor but decreased by wortmannin, indicating that cFLIP expression is regulated by both the NF-κB and phosphatidylinostiol-3 kinase (PI-3)/Akt pathways. These results suggest that cFLIP plays an important role in cell survival not simply by inhibiting death-receptor-mediated apoptosis but also by regulating NF-κB activation in human HCCs.
REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 15 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:515081 HCAPLUS
DOCUMENT NUMBER: 139:147497
TITLE: Over-expression of Smac promotes TRAIL-induced cell death in human hepatocellular carcinoma
AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; **Inoue,**

Hidekazu; Kawakita, Tomoyuki; Saitou, Yukiko; Enokimura, Naoyuki; Yamamoto, Norihiko; Sugimoto, Kazushi; Fujikawa, Katsuhiko; Murata, Kazumoto; Nakano, Takeshi

CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, 514-8507, Japan

SOURCE: International Journal of Molecular Medicine (2003), 12(1), 25-28
CODEN: IJMMFG; ISSN: 1107-3756

PUBLISHER: International Journal of Molecular Medicine

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The second mitochondria-derived activator of caspase, Smac, is an apoptosis-related protein. Smac releases inhibition of the IAP family from caspase-3 to induce apoptosis. Smac is expressed in some malignant tumor cells and is released from mitochondria into the cytosol after death receptor stimulation to promote apoptosis of tumor cells. In this study, the authors found down-regulated Smac protein expression in hepatocellular carcinoma (HCC) tissues, compared to that in non-tumor hepatic tissues. Simultaneously, caspase-3 expression also decreased in HCC tissues. HCC cell lines did not undergo apoptosis after TRAIL stimulation, although Smac was expressed in these HCC cells. Ectopic Smac alone did not induce cell death, but could sensitize HCC cells to TRAIL stimulation. With over-expression of Smac in HCC cells, TRAIL induced by 10% HCC cell death. The role of Smac in apoptosis signaling pathway in HCC cells warrants further study.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 16 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:461802 HCAPLUS

DOCUMENT NUMBER: 139:261544

TITLE: Artificial model for cystathionine β-synthase: efficient β-replacement reaction with thiols employing a novel pyridoxal model compound having an imidazole function

AUTHOR(S): Miyashita, Kazuyuki; **Murafuji, Hidenobu**; Iwaki, Hiroshi; Yoshioka, Eito; Imanishi, Takeshi

CORPORATE SOURCE: Graduate School of Pharmaceutical Sciences, Osaka University, Suita, Osaka, 565-0871, Japan

SOURCE: Tetrahedron (2003), 59(26), 4873-4879
CODEN: TETRAB; ISSN: 0040-4020

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

OTHER SOURCE(S): CASREACT 139:261544

AB As a second-generation pyridoxal model compound for cystathionine β-synthase, we designed a novel model compound having an ionophore function and an imidazole function and applied it to the β-replacement reaction of various thiols to smoothly give S-substituted cysteines. Peptides having an serine-O-carbonate residue at the N-terminal position were also converted to the corresponding peptides having an S-substituted cysteine residue under the catalytic conditions of the novel pyridoxal model compound

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 17 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:461801 HCAPLUS

DOCUMENT NUMBER: 139:261529

TITLE: Artificial model for cystathionine β-synthase: construction of a catalytic cycle with a pyridoxal model compound having an ionophore function
AUTHOR(S): Miyashita, Kazuyuki; **Murafuji, Hidenobu**; Iwaki, Hiroshi; Yoshioka, Eito; Imanishi, Takeshi
CORPORATE SOURCE: Graduate School of Pharmaceutical Sciences, Osaka University, Suita, Osaka, 565-0871, Japan
SOURCE: Tetrahedron (2003), 59(26), 4867-4872
CODEN: TETRAB; ISSN: 0040-4020
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 139:261529
AB Catalytic transformation of serine-O-carbonate to S-aryl cysteine derivs. was successfully achieved in the presence of Li+ by the use of a pyridoxal model compound having an ionophore function, which is the first example mimicking cystathionine β-synthase, artificially.
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 18 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:260328 HCAPLUS
TITLE: Baseplate processing system, baseplate central processing unit management method, baseplate central processing unit, program and recording media [Machine Translation].
INVENTOR(S): Kamei, Kenji; Kitamoto, Toru; Hamada, Tetsuya; **Inoue, Hidekazu**
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003100577	A2	20030404	JP 2001-290973	20010925
PRIORITY APPLN. INFO.:			JP 2001-290973	20010925

AB [Machine Translation of Descriptors]. The technology which can offer the support contents which are necessary for the user quickly efficiently is offered. As information of device constitution of the baseplate central processing unit 1 which is arranged in baseplate disposal factory 4 of the user is accumulated to information storage server 2 automatically, when at support center 5 information of support was offered, whether or not baseplate central processing unit 1 is the support object from information of the component and the above-mentioned device constitution which are described to the information of support is judged automatically, when it is the support object only, the support contents are transmitted to baseplate disposal factory 4. Therefore, in every user, or can the person in charge of support, exclude the time which closely examines the device constitution which differs every device one by one without being conscious of device constitution completely information of support simply just is inputted to support computer 3, it is possible to offer only the support contents which are necessary for the user quickly efficiently.

L24 ANSWER 19 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:217545 HCAPLUS
TITLE: Baseplate processing system, baseplate central

processing unit management method, baseplate central processing unit, program and recording media. [Machine Translation].
INVENTOR(S): Hamada, Tetsuya; Kamei, Kenji; **Inoue, Hidekazu**; Kitamoto, Toru
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 2003086479	A2	20030320	JP 2001-271599		20010907
TW 224354	B1	20041121	TW 2002-91120376		20020905
CN 1404102	A	20030319	CN 2002-142579		20020906
PRIORITY APPLN. INFO.:			JP 2001-270584	A	20010906
			JP 2001-270699	A	20010906
			JP 2001-271369	A	20010907
			JP 2001-271599	A	20010907

AB [Machine Translation of Descriptors]. The technology which can manage the degree of consumption of the part of the baseplate central processing unit efficiently is offered. Degree of consumption of the part of baseplate central processing unit 1 using the particular part by timing the period of use of the particular part due to timer 117, or with counter 118, is measured by calculation doing the baseplate processing quantity. Degree of consumption of the part which is measured is accumulated to the hard disk 24 of information storage server 2 as information 241 of degree of consumption. Whether or not information 241 of degree of consumption is acquired from support computer, 3 has been above the specified value to which degree of consumption is beforehand set every part, as is decided, when it is above specified value, gives out warning the effect where warning occurrence section 313 urges the replacement of the particular part, the order signal concerning the new part which order signal transmission section 314 should exchange with the particular part in incoming order server 8 of part center 7 is transmitted.

L24 ANSWER 20 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:210433 HCAPLUS
TITLE: Baseplate processing system. [Machine Translation].
INVENTOR(S): Hamada, Tetsuya; Kamei, Kenji; **Inoue, Hidekazu**; Kitamoto, Toru
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 2003080155	A2	20030318	JP 2001-271369		20010907
TW 224354	B1	20041121	TW 2002-91120376		20020905
CN 1404102	A	20030319	CN 2002-142579		20020906
PRIORITY APPLN. INFO.:			JP 2001-270584	A	20010906
			JP 2001-270699	A	20010906
			JP 2001-271369	A	20010907

JP 2001-271599 A 20010907

AB [Machine Translation of Descriptors]. The baseplate processing system which can do operation education efficiently in the operator is offered. Plural baseplate central processing units 1 are arranged in baseplate disposal factory 4. In addition, support computer 3 is arranged in support center, 5 support computer 3 and plural baseplate central processing units 1 at network (Internet) is connected. The education 341 program is housed in the fixed 34 disk which 3 support computers of 5 support centers have. CPU31 of 3 support computers reading out this education 341 program, transmission section 315 it is possible by executing, to transmit the information of the education regarding the operation of the said device in each baseplate central processing unit 1 of baseplate disposal factory 4 via network from Communication Div. 38.

L24 ANSWER 21 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:201167 HCAPLUS
TITLE: Baseplate central processing unit management system and baseplate central processing unit, baseplate central processing unit management method, program, and recording media. [Machine Translation].
INVENTOR(S): Hamada, Tetsuya; Kamei, Kenji; **Inoue, Hidekazu**; Kitamoto, Toru
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003077822	A2	20030314	JP 2001-270584	20010906
TW 224354	B1	20041121	TW 2002-91120376	20020905
CN 1404102	A	20030319	CN 2002-142579	20020906

PRIORITY APPLN. INFO.:

JP 2001-270584	A	20010906
JP 2001-270699	A	20010906
JP 2001-271369	A	20010907
JP 2001-271599	A	20010907

AB [Machine Translation of Descriptors]. While making the job burden of the operator lighten, initial setting operation of the baseplate central processing unit easy, at the same time, it designates that the network system which is done securely is offered as topic. As for baseplate central processing unit 1, early operation is decided by control supervisor 152 and device basic data 151b. Support computer 3 has accumulated the device basic data 151b which is managed in every version of control supervisor 152. Basic data required section 121 of baseplate central processing unit 1 forwards the request-to-send of the basic data vis-a-vis the basic data setting section 321 of support computer 3. In this case, version of the control supervisor 152 which is acquired with version acquisition section 122 is together notified. Because of this, basic data setting section 321 transmits the device basic data 151b which corresponds to version to baseplate central processing unit 1. In baseplate central processing unit 1, the device basic data 151b which is received with basic data register section 123, is registered.

L24 ANSWER 22 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:201142 HCAPLUS
TITLE: Baseplate central processing unit management system and baseplate central processing unit, baseplate

central processing unit management method, program, and recording media. [Machine Translation].
INVENTOR(S): Hamada, Tetsuya; Kamei, Kenji; **Inoue, Hidekazu**; Kitamoto, Toru
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 13 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003077787	A2	20030314	JP 2001-270699	20010906
TW 224354	B1	20041121	TW 2002-91120376	20020905
CN 1404102	A	20030319	CN 2002-142579	20020906
PRIORITY APPLN. INFO.:			JP 2001-270584 A	20010906
			JP 2001-270699 A	20010906
			JP 2001-271369 A	20010907
			JP 2001-271599 A	20010907

AB [Machine Translation of Descriptors]. It designates that information in order to control the operation of the baseplate central processing unit without applying burden on the user, is backed up efficiently as topic. Information 151 of setting, control supervisor 152,153 is housed in memory section 104,114 of baseplate central processing unit 1, as for baseplate central processing unit 1, following to these items of information and the program, operation is controlled. Local indication section 121 to have schedule function, schedule therefore indication order of backup is forwarded. Responding to this indication order, duplication information acquisition section 122 was housed in memory section 104,114 to form the duplication of the information which is appointed, information of duplication through network, is transferred to the housing section 221 of information storage server 2. It houses housing section 221, in hard disk 24 with the information of the duplication which is received as a backup data 251. In addition, housing section 221 housing only the finite difference data of information of duplication is possible.

L24 ANSWER 23 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:126370 HCAPLUS
DOCUMENT NUMBER: 138:276622
TITLE: Adjustment of Perdew-Wang exchange functional for describing van der Waals and DNA base-stacking interactions
AUTHOR(S): Kurita, Noriyuki; **Inoue, Hidekazu**; Sekino, Hideo
CORPORATE SOURCE: Department of Knowledge-Based Information Engineering, Toyohashi University of Technology, Toyohashi, Aichi, 441-8580, Japan
SOURCE: Chemical Physics Letters (2003), 370(1,2), 161-169
CODEN: CHPLBC; ISSN: 0009-2614
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In order to accurately describe the van der Waals interaction between rare-gas atoms by the d. functional theory, we adjusted the exchange-functional developed by Perdew and Wang (PW). The van der Waals interactions of He, Ne, Ar and Kr dimers were investigated. The adjustment improves the overestimation of the interactions by the original PW exchange-functional, providing the qual. accurate trend in van der

Waals interactions of He, Ne, Ar and Kr dimers. The adjusted functional for He and Ne underestimates the DNA base-stacking interaction between cytosine monomers. This may indicate that the PW exchange-functional requires a further modification or a van der Waals correction in order to give accurate DNA base-stacking interaction.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 24 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:57030 HCAPLUS
TITLE: Baseplate processing system and baseplate central processing unit, additional information acquisition method, program and recording media. [Machine Translation].
INVENTOR(S): Kitamoto, Toru; Kamei, Kenji; **Inoue, Hidekazu**; Hamada, Tetsuya
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 2003022200	A2	20030124	JP 2001-205111		20010705
CN 1396625	A	20030212	CN 2002-130372		20020705
PRIORITY APPLN. INFO.:			JP 2001-205109	A	20010705
			JP 2001-205110	A	20010705
			JP 2001-205111	A	20010705

AB [Machine Translation of Descriptors]. The time of obstacle of the baseplate central processing unit, the baseplate processing system which can acquire the information of up-to-date coping instantaneously is offered. Baseplate processing system 10 baseplate central processing unit has with 1 and support computer 3, is connected to the respective network 6. Information 363 of up-to-date coping is accumulated to support computer 3, by the system manager. When obstacle occurs with baseplate central processing unit 1, following to alarm defined file 161 with alarm process division, 122 as you can do the control of operation, contents of obstacle are indicated in display part 130. Furthermore, information 363 of up-to-date coping where coping information acquisition section 128 corresponds to particular obstacle in support computer 3 is required. Information 363 of up-to-date coping replies from coping information dissemination section 324 of support computer 3 in consequence of this. Because of this, it can peruse the information of up-to-date coping instantaneously in the time of obstacle.

L24 ANSWER 25 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:57029 HCAPLUS
TITLE: Baseplate processing system, baseplate central processing unit management method, baseplate central processing unit, program and recording media. [Machine Translation].
INVENTOR(S): Kitamoto, Toru; Kamei, Kenji; **Inoue, Hidekazu**; Hamada, Tetsuya
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 18 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent

LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 2003022188	A2	20030124	JP 2001-205110		20010705
CN 1396625	A	20030212	CN 2002-130372		20020705
PRIORITY APPLN. INFO.:			JP 2001-205109	A	20010705
			JP 2001-205110	A	20010705
			JP 2001-205111	A	20010705

AB [Machine Translation of Descriptors]. When software module is installed to the baseplate central processing unit, the baseplate processing system which can prevent the obstacle which originates in the unconformity between each software module beforehand is offered. When system control section 100 of a certain baseplate central processing unit 1 or software module 128 is installed to unit control section 115, accumulating the version information of the software module 128 where system control section 100 at that point in time and unit control section 115 is installed respectively, it constructs version management table 241. Consistent verification section 235 while referring to the verification table 242 which, registers the version information of the software module 128 which possesses consistency mutually verifies the consistency between each software module from version management table 241.

L24 ANSWER 26 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:57025 HCAPLUS
TITLE: Baseplate processing system and baseplate central processing unit, device information management method, program and recording media. [Machine Translation].
INVENTOR(S): Kitamoto, Toru; Kamei, Kenji; **Inoue, Hidekazu**; Hamada, Tetsuya
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 17 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 2003022116	A2	20030124	JP 2001-205109		20010705
CN 1396625	A	20030212	CN 2002-130372		20020705
PRIORITY APPLN. INFO.:			JP 2001-205109	A	20010705
			JP 2001-205110	A	20010705
			JP 2001-205111	A	20010705

AB [Machine Translation of Descriptors]. The time of obstacle of the baseplate central processing unit, immediately can peruse information of work from remote place the baseplate processing system which is offered. Baseplate processing system 10 baseplate central processing unit 1 and information storage server has 2 and support computer 3, is connected to the respective network 6. When obstacle occurs with baseplate central processing unit 1, as for alarm process division 122 extracting necessary related log file 262, you remember in hard disk 24 of information storage server 2. Furthermore, obstacle information is formed by obstacle information formation department, 123 finally is remembered in hard disk 24 of information storage server 2 as obstacle information DB261. As for these related log files 262 and obstacle information DB261, it becomes possible to peruse from support computer 3 of remote place with device

opening of information section 226. Because of this, related log file it can peruse 262 and obstacle information DB261 instantaneously.

L24 ANSWER 27 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:706835 HCAPLUS
DOCUMENT NUMBER: 138:104598
TITLE: Functional Expression of Tumor Necrosis Factor-Related Apoptosis-Inducing Ligand in Human Colonic Adenocarcinoma Cells
AUTHOR(S): **Inoue, Hidekazu**; Shiraki, Katsuya; Yamanaka, Takenari; Ohmori, Shigeru; Sakai, Takahisa; Deguchi, Masatoshi; Okano, Hiroshi; Murata, Kazumoto; Sugimoto, Kazushi; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, Mie, Japan
SOURCE: Laboratory Investigation (2002), 82(9), 1111-1119
CODEN: LAINAW; ISSN: 0023-6837
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB TNF-related apoptosis-inducing ligand (TRAIL) can induce apoptosis in various transformed cell lines. Therefore, we investigated TRAIL sensitivity, TRAIL-induced nuclear factor-κB (NF-κB) activation, and expression of TRAIL in human colonic adenocarcinoma cell lines (HT-29, LS180, SK-CO-1). All four TRAIL receptors (TRAIL-R1 through TRAIL-R4) are expressed in these cell lines. TRAIL sensitivity was assessed by assay of cell viability. Cancer cell viabilities were 83 ± 3.1% (HT-29), 90 ± 4.3% (LS180), and 88 ± 6.3% (SK-CO-1) at 24 h after the addition of 100 ng/mL TRAIL, indicating that these cell lines were relatively resistant to TRAIL. Activation of NF-κB was variably influenced by TRAIL administration, with no consistent tendency among the cell lines, indicating that TRAIL-induced NF-κB activation might be cell-type dependent. In contrast, TRAIL was expressed in the human colonic adenocarcinoma cell lines by Western blotting and RT-PCR. Increased expression of TRAIL on tumor cells was observed by flow cytometry after cytokine stimulation (IFN-γ, TNF-α) or the addition of chemotherapeutic agents (camptothecin, doxorubicin hydrochloride). TRAIL on HT-29 cells was functional and able to induce apoptosis in Jurkat cells. Jurkat cell viability was increased by the addition of TRAILR1-R4-Fc. In the presence of various cytokines or chemotherapeutic agents, functional TRAIL is expressed on the surface of tumor cells, and this expressed TRAIL might contribute to tumor immune privilege by inducing apoptosis of activated human lymphocytes.
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 28 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:694512 HCAPLUS
DOCUMENT NUMBER: 138:252460
TITLE: Peroxisome proliferator-activated receptor γ augments tumor necrosis factor family-induced apoptosis in hepatocellular carcinoma
AUTHOR(S): Okano, Hiroshi; Shiraki, Katsuya; **Inoue, Hidekazu**; Yamanaka, Takenari; Deguchi, Masatoshi; Sugimoto, Kazushi; Sakai, Takahisa; Ohmori, Shigeru; Fujikawa, Katsuhiko; Murata, Kazumoto; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, 514-8507, Japan
SOURCE: Anti-Cancer Drugs (2002), 13(1), 59-65
CODEN: ANTDEV; ISSN: 0959-4973

PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Proliferator-activated receptor γ (PPARγ) is a nuclear receptor, which mainly assocs. with adipogenesis, but also appears to facilitate cell differentiation or apoptosis in certain malignant cells. This apoptosis induction by PPARγ is increased by co-stimulation with tumor necrosis factor (TNF)-α-related apoptosis-inducing ligand (TRAIL), a member of the TNF family. In this study, we investigated the effect of PPARγ on Fas-mediated apoptosis in hepatocellular carcinoma (HCC) cell lines. PPARγ was expressed on all seven HCC cell lines and located in their nuclei. 15-Deoxy-Δ-12,14-prostaglandin J2 (15d-PGJ2), a PPARγ ligand, inhibited cellular proliferation in HepG2, SK-Hep1 or HLE cells, unlike pioglitazone, another PPARγ ligand, which did not have a significant influence on proliferation of these cells. However, 15d-PGJ2 facilitated Fas-mediated HCC apoptosis that could not be induced by Fas alone. These results suggest that PPARγ can augment TNF-family-induced apoptosis.
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 29 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:665453 HCAPLUS
DOCUMENT NUMBER: 138:22971
TITLE: Expression of survivin during liver regeneration
AUTHOR(S): Deguchi, Masatoshi; Shiraki, Katsuya; **Inoue, Hidekazu**; Okano, Hiroshi; Ito, Takeshi; Yamanaka, Takenari; Sugimoto, Kazushi; Sakai, Takahisa; Ohmori, Shigeru; Murata, Kazumoto; Furusaka, Akihiro; Hisatomi, Hisashi; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, Mie, 2-174, Japan
SOURCE: Biochemical and Biophysical Research Communications (2002), 297(1), 59-64
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Survivin functions to suppress cell death and regulate cell division, and is observed uniquely in tumor cells and developmental cells. However, the expression and regulation of survivin in non-transformed cells are not well elucidated. Therefore, we investigated the expression of survivin in a murine liver regeneration model after partial hepatectomy and i.p. carbon tetrachloride (CCl4) injection. We found that the expression of survivin transcript and protein were markedly elevated with the onset of DNA synthesis and remained elevated during G2 and M phases during liver regeneration. In a normal mouse liver cell line, over-expression of survivin resulted in a decrease in the G0/G1 phase and an increase in the S and G2/M phases, resulting in Rb phosphorylation. These findings suggest that survivin is dramatically expressed in a cell cycle-dependent manner during liver regeneration and provide a new insight into the regulation of cell proliferation and differentiation.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 30 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:647458 HCAPLUS
DOCUMENT NUMBER: 138:122843
TITLE: β-Replacement reaction of serine-O-carbonate derivatives with thiols catalyzed by a pyridoxal model

having an ionophore side-chain
AUTHOR(S): Miyashita, Kazuyuki; **Murafuji, Hidenobu**; Iwaki, Hiroshi; Yoshioka, Eito; Imanishi, Takeshi
CORPORATE SOURCE: Graduate School of Pharmaceutical Sciences, Osaka University, Suita, Osaka, 565-0871, Japan
SOURCE: Chemical Communications (Cambridge, United Kingdom) (2002), (17), 1922-1923
CODEN: CHCOFS; ISSN: 1359-7345
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 138:122843
AB Serine-O-carbonate derivs., including peptides having a serine-O-carbonate residue at the N-terminal position, are catalytically transformed into S-substituted cysteine derivs. employing the pyridoxal model having an ionophore function in the presence of Li+; this is the first artificial model mimicking cystathionine β-synthase.
REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 31 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:622328 HCAPLUS
DOCUMENT NUMBER: 137:184210
TITLE: Efficacy of long-term interferon therapy in chronic hepatitis B patients with HBV genotype C
AUTHOR(S): Sakai, Takahisa; Shiraki, Katsuya; **Inoue, Hidekazu**; Okano, Hiroshi; Deguchi, Masatoshi; Sugimoto, Kazushi; Ohmori, Shigeru; Murata, Kazumoto; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, Mie, 514-8507, Japan
SOURCE: International Journal of Molecular Medicine (2002), 10(2), 201-204
CODEN: IJMMFG; ISSN: 1107-3756
PUBLISHER: International Journal of Molecular Medicine
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Infection with Hepatitis B virus (HBV) genotype C predominates in Japan. We analyzed the efficacy of interferon (IFN) α or β in the treatment of chronic hepatitis B patients with HBV genotype C and the clin. predictors for therapeutic response. Forty-three genotype C-infected, chronic hepatitis B e antigen (HBeAg)-pos. patients (32 men and 11 women with a mean age of 35.6±10.1 yr) who had been treated with IFN therapy were retrospectively studied. The patients were classified into two treatment groups. Short-term therapy group was administered a 5-6 MU dose three times weekly for 4 wk, and the long-term therapy group for 24 wk. At the end of the follow-up period, 4 (15%) of 27 short-term therapy group patients and 6 (38%) of 16 long-term therapy group patients had normalized serum ALT levels and sero-conversion of HBeAg to anti-HBe (p=0.137). Multivariate anal. for parameters most important for the efficacy of IFN therapy was performed using Cox proportional hazard models in order to investigate the association between baseline characteristics of patients and the response to IFN treatment. As a result, the p-values of IFN treatment group and sex were <0.05, and both factors can be recognized as independent significant factors (relative risk, 2.93 and 2.53; p=0.027 and 0.040, resp.). Furthermore, the cumulative rates of seroconversion of HBeAg to anti-HBe analyzed by the Kaplan-Meier method was significantly higher in the female group (p=0.015) and in the long-term IFN therapy group (p=0.0046). In summary, long-term IFN therapy may be more effective than short-term IFN therapy for patients with chronic HBV genotype C

infection.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 32 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:366480 HCAPLUS
DOCUMENT NUMBER: 137:304390
TITLE: Histone deacetylase inhibitors sensitize human colonic adenocarcinoma cell lines to TNF-related apoptosis inducing ligand-mediated apoptosis
AUTHOR(S): **Inoue, Hidekazu**; Shiraki, Katsuya; Ohmori, Shigeru; Sakai, Takahisa; Deguchi, Masatoshi; Yamanaka, Takenari; Okano, Hiroshi; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Mie, 514-8507, Japan
SOURCE: International Journal of Molecular Medicine (2002), 9(5), 521-525
CODEN: IJMMFG; ISSN: 1107-3756
PUBLISHER: International Journal of Molecular Medicine
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Histone deacetylase inhibitor (HDAI) induces accumulation of highly acetylated histones by inhibiting the activity of histone deacetylase and inhibits cell proliferation, induces differentiation, and promotes apoptosis. TNF-related apoptosis inducing ligand (TRAIL) induces apoptosis in various human cancer cells, a promising observation because it raises the possibility of a death ligand selective for tumor cells. However, resistance to TRAIL-induced apoptosis was seen in colonic adenocarcinoma cell lines. So we investigated whether human colonic adenocarcinoma cell lines can be sensitized to TRAIL-induced apoptosis by the addition of HDAI. We investigated sensitivity to histone deacetylase inhibitor in colonic adenocarcinoma cell lines using the MTT assay. Cell viability decreased with sodium butyrate (SB) and trichostatin A (TSA) in a dose-dependent manner in LS 180 and HT-29 cells. Nuclear condensation and fragmentation were observed by DAPI staining after 24 h stimulation with SB or TSA in LS 180 cells. We also investigated the combination of HDAI and TNF family members (TRAIL, anti-Fas antibody or TNFα) in colonic adenocarcinoma cell lines. HDAI augmented TNF family-related apoptosis in LS 180 cells and HT-29 cells. HDAI sensitizes human colonic adenocarcinoma cell lines to TRAIL-mediated apoptosis. Thus, HDAI may be useful as an adjuvant agent for TRAIL in the treatment of human colonic adenocarcinomas that are resistant to TRAIL.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 33 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:502211 HCAPLUS
DOCUMENT NUMBER: 135:257445
TITLE: Regio- and stereoselective α-alkylation of N-terminal amino acid residue of peptides using a pyridoxal model compound with a chiral ansa-structure
AUTHOR(S): Miyashita, Kazuyuki; Iwaki, Hiroshi; Tai, Kuninori; **Murafuji, Hidenobu**; Sasaki, Naoko; Imanishi, Takeshi
CORPORATE SOURCE: Graduate School of Pharmaceutical Sciences, Osaka University, Suita, Osaka, 565-0871, Japan
SOURCE: Tetrahedron (2001), 57(27), 5773-5780
CODEN: TETRAB; ISSN: 0040-4020
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal

LANGUAGE: English
OTHER SOURCE(S): CASREACT 135:257445
AB Regio- and stereoselective α-alkylation of N-terminal amino acid residue of peptides was achieved by Li+-mediated alkylation of aldimines prepared from the peptides and a pyridoxal model compound having a chiral ansa-structure and an ethoxyethoxy group at C-3. The stereochem. and stereoselectivity of the reaction were found to be influenced predominantly by the chirality of the model compound and Li+, but little by the stereochem. of the original peptides.
REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 34 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:293230 HCAPLUS
DOCUMENT NUMBER: 135:56038
TITLE: Discovery of a non-peptide small molecule that selectively mimics the biological actions of calcitonin
AUTHOR(S): Katayama, T.; Furuya, M.; Yamaichi, K.; Konishi, K.; Sugiura, N.; **Murafuji, H.**; Magota, K.; Saito, M.; Tanaka, S.; Oikawa, S.
CORPORATE SOURCE: Suntory Biomedical Research Limited, Shimamoto-cho, Mishima-gun, Osaka, 618-8503, Japan
SOURCE: Biochimica et Biophysica Acta, General Subjects (2001), 1526(2), 183-190
CODEN: BBGSB3; ISSN: 0304-4165
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Calcitonin (CT), a 32-amino acid peptide hormone secreted mainly from the thyroid gland, plays an important role in maintaining bone homeostasis. To discover non-peptide small mols. with biol. actions similar to those of CT, a cell-based screening of an inhouse chemical library was performed and a pyridone derivative (SUN B8155) was identified. Like CT, it elevated cAMP levels in T47D and UMR106-06 cells which endogenously express human and rat CT receptor, resp. SUN B8155 also stimulated cAMP formation in cells expressing recombinant human CT receptor, but not in those expressing human parathyroid hormone/parathyroid hormone-related peptide receptor. Accumulation of cAMP in T47D cells was blocked by a selective antagonist of CT receptor, salmon CT(8-32), whereas SUN B8155 did not displace the specific binding of [125I]CT to the receptor. Our results suggested that the compound selectively interacts with the CT receptor by a mechanism similar to but probably different from that of CT itself. In rats, i.p. administration of SUN B8155 significantly lowered serum calcium levels, like CT. Our results demonstrate, for the first time, that the biol. activities of the newly identified small mol. can mimic that of CT, acting via the CT receptor.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 35 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:164313 HCAPLUS
DOCUMENT NUMBER: 135:14048
TITLE: Identification of a Novel Inhibitor of LPS-Induced TNF-α Production with Antiproliferative Activity in Monocyte/Macrophages
AUTHOR(S): Nagahira, Asako; Nagahira, Kazuhiro; **Murafuji, Hidenobu**; Abe, Keiichi; Magota, Koji; Matsui, Masashi; Oikawa, Shinzo
CORPORATE SOURCE: Suntory Biomedical Research Limited, Mishima-gun,

Osaka, 618-8503, Japan
SOURCE: Biochemical and Biophysical Research Communications (2001), 281(4), 1030-1036
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An isoquinoline derivative, 5-methyl-7,8-dimethoxy-1-phenylpyrazolo[5,4-c]isoquinoline (compound 1), was identified as a novel inhibitor of LPS-induced TNF-α production by cell-based screening. Compound 1 suppressed LPS-induced TNF-α production in RAW264.7 cells and murine peritoneal macrophages in a dose-dependent manner similar to SB203580, known as a specific inhibitor of p38 MAPK. It also inhibited an LPS-induced increase in serum TNF-α in a mouse endotoxic shock model with an ED50 of .apprx.10 mg/kg. Compound 1 had little effect on the incorporation of [3H]-leucine into the cells, while it suppressed LPS-induced TNF-α mRNA levels in RAW264.7 cells. The results indicate that suppression of TNF-α production was not a result of nonspecific inhibition of de novo translation but was based on the decreased TNF-α mRNA levels. The in vitro kinase assay revealed that compound 1 did not strongly inhibit p38 MAPK activity, its potency being much lower than that of SB203580, suggesting that the TNF-α-suppressive action of compound 1 cannot be attributed to the inhibition of p38 MAPK. Furthermore, in contrast to SB203580, it significantly inhibited the growth of RAW264.7 and THP-1 cells in a cytostatic manner. Compound 1 is likely to have antiinflammatory and antiproliferative effects by acting on some mol. other than p38 MAPK that contributes to both LPS-induced TNF-α production and the cell growth of monocyte/macrophages. (c) 2001 Academic Press.
REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 36 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:805716 HCAPLUS
DOCUMENT NUMBER: 134:220593
TITLE: Effect of segmental transcatheter arterial chemoembolization on branched chain amino acids and tyrosine ratio in patients with hepatocellular carcinoma
AUTHOR(S): **Inoue, Hidekazu**; Ito, Takeshi; Siraki, Katsuya; Sugimoto, Kazushi; Sakai, Takahisa; Oomori, Shigeru; Takase, Koujirou; Nakano, Takeshi
CORPORATE SOURCE: First Department of Internal Medicine, Mie University School of Medicine, Tsu, Mie, 514-8507, Japan
SOURCE: International Journal of Oncology (2000), 17(5), 977-980
CODEN: IJONES; ISSN: 1019-6439
PUBLISHER: International Journal of Oncology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of segmental transcatheter arterial chemoembolization (TAE) on serum amino acid levels and liver function were studied in 23 patients with HCC associated with hepatitis virus C (22 patients) or alcoholism (1 patient), with compensated liver cirrhosis (child A 18 patients, child B 5 patients). Blood serum levels of branched-chain amino acids (BCAA), Tyr, branched-chain amino acids to Tyr ratio (BTR), ammonia, total bilirubin and albumin, and prothrombin times were measured before and after TAE (24 h, 7 and 14 days). The BTR was increased significantly 24 h after TAE ($p<0.001$) and gradually decreased to pre-TAE levels. Serum Tyr levels decreased at 24 h after TAE ($p<0.005$) and later increased. Serum BCAA

levels increased slightly at 7 days after TAE and were decreased at 14 days after TAE. This results indicated that the increased BTR was due primarily to the decreased Tyr level at 24 h after TAE. Serum ammonia levels gradually decreased after TAE and the prothrombin time and serum levels of total bilirubin and albumin were not significantly changed. In this study, segmental TAE had little influence on liver function, and the BTR unexpectedly increased at 24 h after TAE. These results suggest that segmental TAE has minimal side effects and may have a beneficial effect on amino acid metabolism

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 37 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:741689 HCAPLUS
TITLE: The substrate central processing unit and the recording media which records the control program. [Machine Translation].
INVENTOR(S): Murata, Kinya; **Inoue, Hidekazu**; Yoshida, [NAME NOT TRANSLATED]; Kamei, Kenji
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 11 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000294483	A2	20001020	JP 1999-96395	19990402
JP 3631041	B2	20050323		
PRIORITY APPLN. INFO.:			JP 1999-96395	19990402

AB [Machine Translation of Descriptors]. It is to offer the baseplate central processing unit where input error of treatment contents is prevented and the memory medium which remembers its control program. Inline controller 1 in main controller with the Vs flow information receives the treatment information which is set 3 and exposure machine 20. In when starting the treatment, the treatment information is indicated in the picture of inline panel 2, the treatment information which is selected by selecting which of the treatment information where the operator is indicated is inputted. Inline controller 1 gives the baseplate turnaround time which is received from exposure machine 20 to main controller 3. As for main controller 3 baseplate turnaround time with of process division 5 is decided on the basis of the baseplate turnaround time which is given. Inline controller 1 starts treatment in main controller 3 and exposure machine 20 on the basis of the treatment information and the Vs flow information which are inputted consecutively the directive.

L24 ANSWER 38 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:666367 HCAPLUS
TITLE: The substrate central processing unit and the recording media which records the communication control program. [Machine Translation].
INVENTOR(S): Kitamoto, Toru; **Inoue, Hidekazu**; Yoshida, [NAME NOT TRANSLATED]
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 9 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000260675	A2	20000922	JP 1999-66235	19990312

PRIORITY APPLN. INFO.: JP 1999-66235 19990312

AB [Machine Translation of Descriptors]. It is possible to operate the insertion and deletion of the message being retained when losing of communication with the computer easily it is to offer the baseplate central processing unit which has the communicator talent where the influence which damage occurs in the disk record of part and gives to other message is little. When losing of communication with the host computer 30, the message which should transmit to host computer 30 through data base management software 230, houses the communication control module 210 of main R trawler 20, in the spool table which is formed by data base 240 at the time of the retrieval of communication with the host computer 30, transmits the message which is housed in the spool table to host computer 30.

L24 ANSWER 39 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:663800 HCAPLUS
TITLE: The substrate central processing unit and the recording media which records the communication control program. [Machine Translation].
INVENTOR(S): Kitamoto, Toru; **Inoue, Hidekazu**; Yoshida, [NAME NOT TRANSLATED]
PATENT ASSIGNEE(S): Dainippon Screen Mfg. Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 11 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000260676	A2	20000922	JP 1999-66236	19990312

PRIORITY APPLN. INFO.: JP 1999-66236 19990312

AB [Machine Translation of Descriptors]. It is to offer the baseplate central processing unit which has the communicator talent which can do the modification of variable easily at the time of communicating with the host computer. Device control module 220 of main R trawler 20, when the event occurs from slave controller 21, transfers the event and port number to communication control module 210, is worthy of the variable which is annexed to the event through data base management software 230, in data base 240 the loading. As for communication control module 210, acquire the value of the variable which the loading is done in data base 240 on the basis of the port number which is given from device control module 220, transmit to host computer 30 with the value of the event and variable as message.

L24 ANSWER 40 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:209233 HCAPLUS
DOCUMENT NUMBER: 133:124907
TITLE: Chemiluminescence determination of iodide and/or iodine using a luminol-hexadecyltrimethylammonium chloride reversed micelle system following on-line oxidation and extraction
AUTHOR(S): Fujiwara, Terufumi; **Inoue, Hidekazu**;

Mohammadzai, Imdad U.; Kumamaru, Takahiro
CORPORATE SOURCE: Dep. Chem., Grad. Sch. Sci., Hiroshima University, Higashi-Hiroshima, 739-8526, Japan
SOURCE: Analyst (Cambridge, United Kingdom) (2000), 125(4), 759-763
CODEN: ANALAO; ISSN: 0003-2654
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A rapid and sensitive flow method, based on the combination of online oxidation-solvent extraction with reversed micellar mediated luminol chemiluminescence detection, was found to be suitable for the determination of iodide in aqueous solution The flow procedure involved the oxidation of iodide to iodine, extraction of iodine into cyclohexane followed by membrane phase separation, and its chemiluminescence detection using the reaction of iodine with luminol in a reversed micellar solution of hexadecyltrimethylammonium chloride in 6 +5 (volume/volume) chloroform-cyclohexane/water (buffered with sodium carbonate). The optimum conditions for iodide oxidation were evaluated using 2-iodosobenzoate as an oxidizing agent and a detection limit of 0.02 ng mL-1 iodide was achieved. Using different oxidants, 2-iodosobenzoate and peroxodisulfate, linear calibration graphs were obtained with dynamic ranges from 5 to 200 and from 50 to 5000 ng mL-1, resp. The proposed method was also applied to a mixture of iodine and iodide, where iodine was determined directly without using an oxidizing agent, total iodine (iodine +iodide) was determined using an oxidizing agent, and iodide was calculated by difference. The method was applied to the differential determination of iodide and iodine in gargle samples with a precision of ca. 4% relative standard deviation.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

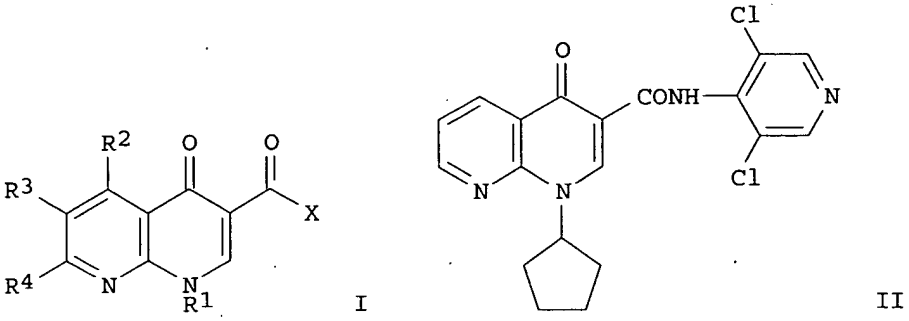
L24 ANSWER 41 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:495295 HCAPLUS
DOCUMENT NUMBER: 131:129983
TITLE: Preparation of 1-cycloalkyl-1,8-naphthyridin-4-one derivatives with phosphodiesterase IV inhibitory activity
INVENTOR(S): Shimamoto, Tetsuo; **Inoue, Hidekazu**; Hayashi, Yasuhiro
PATENT ASSIGNEE(S): Suntory Limited, Japan
SOURCE: PCT Int. Appl., 165 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9938867	A1	19990805	WO 1999-JP404	19990129
W: AU, CA, CN, HU, JP, KR, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2285352	AA	19990805	CA 1999-2285352	19990129
AU 9921856	A1	19990816	AU 1999-21856	19990129
AU 763636	B2	20030731		
EP 978516	A1	20000209	EP 1999-901925	19990129

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

US 6331548 B1 20011218 US 1999-402142 19990929
PRIORITY APPLN. INFO.: JP 1998-17009 A 19980129
WO 1999-JP404 W 19990129
OTHER SOURCE(S): MARPAT 131:129983

GI



I II

AB 1-Cycloalkyl-1,8-naphthyridin-4-one derivs. represented by formula (I) or pharmacol. acceptable salts or solvates thereof (wherein R1 represents optionally substituted cycloalkyl or optionally substituted heterocycloalkyl; R2, R3, and R4 each independently represents hydrogen, optionally substituted lower alkyl, or halogeno; and X represents NR5R6 or OR7 (wherein R5 and R6 each independently represents hydrogen, optionally substituted lower alkyl, optionally substituted cycloalkyl, optionally substituted aryl, or optionally substituted heteroaryl); and R7 represents hydrogen, optionally substituted lower alkyl, or optionally substituted cycloalkyl) are prepared These compds. selectively inhibit phosphodiesterase IV and production of tumor necrosis factor TNF-α and are useful for the prevention and treatment of phosphodiesterase IV-associated diseases such as respiratory disease (bronchial asthma and chronic bronchitis), nerve functional disorders (depression, schizophrenia, Alzheimer's disease or Parkinson's disease-related learning, memory, and cognition disorders), inflammatory diseases (atopic dermatitis, conjunctivitis, or AIDS), general or local joint diseases (knee arthritis deformans and chronic rheumatoid arthritis) and cytokine-associated diseases such as psoriasis, septicemia, Crohn's disease, cardiac infarction, arteriosclerosis, and nephritis. Thus, 1-cyclopentyl-1,4-dihydro-1,8-naphthyridine-4-one-3-carboxylic acid was refluxed with SOCl2 in toluene for 1.5 h, evaporated in vacuo, and then condensed with 4-amino-3,5-dichloropyridine in the presence of NaH in THF to give the title compound, N-(3,5-dichloropyridin-4-yl)-cyclopentyl-1,4-dihydro-1,8-naphthyridine-4-one-3-carboxamide (II). II showed IC50 of 0.0003 μM against phosphodiesterase IV.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 42 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:126900 HCAPLUS
DOCUMENT NUMBER: 130:196577
TITLE: Preparation of 1-aryl-1,8-naphthyridin-4-ones as type IV phosphodiesterase inhibitors
INVENTOR(S): Shimamoto, Tetsuo; **Inoue, Hidekazu**; Hayashi, Yasuhiro
PATENT ASSIGNEE(S): Suntory Limited, Japan

SOURCE: PCT Int. Appl., 112 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9907704	A1	19990218	WO 1998-JP3510	19980806
W: AL, AU, CA, CN, HU, KR, LT, LV, MK, RO, SI, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2268190	AA	19990218	CA 1998-2268190	19980806
AU 9885607	A1	19990301	AU 1998-85607	19980806
AU 755350	B2	20021212		
JP 11106385	A2	19990420	JP 1998-223178	19980806
EP 958297	A1	19991124	EP 1998-936683	19980806
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 6297248	B1	20011002	US 1999-284019	19990406
US 2002006935	A1	20020117	US 2001-907741	20010719
US 6541480	B2	20030401		

PRIORITY APPLN. INFO.: JP 1997-212322 A 19970806
WO 1998-JP3510 W 19980806
US 1999-284019 A3 19990406
OTHER SOURCE(S): MARPAT 130:196577
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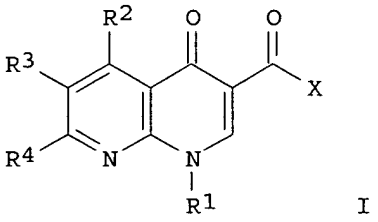
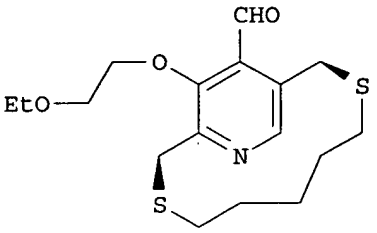


I

AB The title compds. [I; R1 = (un)substituted aryl, heteroaryl; R2-R4 = H, alkyl, halo; X = NR5R6, OR7 (wherein R5, R6 = H, alkyl, cycloalkyl, etc.; R7 = H, alkyl, cycloalkyl)], type IV phosphodiesterase inhibitors, were prepared Thus, reaction of 1-(4-fluorophenyl)-1,4-dihydro[1,8]naphthyridin-4-one-3-carboxylic acid with SOCl2 in THF followed by treatment of a THF solution of the resulting acid chloride with aqueous NH4OH afforded 54% I [R1 = 4-FC6H4; R2-R4 = H; X = NH2] which showed IC50 of 1.40 μM against PDE IV and IC50 of 0.40 μM against TNF-α production
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 43 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:599064 HCAPLUS
DOCUMENT NUMBER: 130:52708
TITLE: Stereoselective and N-terminal selective α-alkylation of peptides using a pyridoxal model compound as a chiral N-terminal activator
AUTHOR(S): Miyashita, Kazuyuki; Iwaki, Hiroshi; Tai, Kuninori; **Murafuji, Hidenobu**; Imanishi, Takeshi

CORPORATE SOURCE: Graduate School of Pharmaceutical Sciences, Osaka University, Osaka, 565-0871, Japan
SOURCE: Chemical Communications (Cambridge) (1998), (18), 1987-1988
CODEN: CHCOFS; ISSN: 1359-7345
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 130:52708
GI



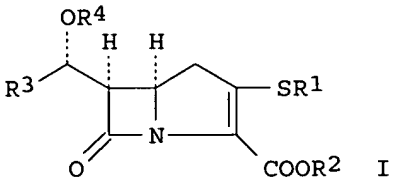
I

AB Stereoselective and N-terminal selective α-alkylation of peptides is achieved using a pyridoxal model compound I as an N-terminal activator which also functions as a chiral auxiliary. Reaction of the aldimines prepared from pyridoxal compound I and peptides e.g. (H-Ala-Ala-OCH2Ph, H-Ala-D-Ala-OCH2Ph, H-Ala-Val-OCH2Ph) with alkyl bromides in the presence of LiClO4 and DBU stereoselectively afforded N-terminal α-alkylated peptides after an acidic treatment. The reaction without Li+ or with other alkali metal ions showed the reverse stereoselectivity.
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 44 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:143389 HCAPLUS
DOCUMENT NUMBER: 128:243874
TITLE: Preparation of carbapenems, bactericides containing them, and their intermediates
INVENTOR(S): Ishgkuro, Masamichi; Nakatsuka, Takashi; **Inoue, Hidekazu**
PATENT ASSIGNEE(S): Suntory, Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 34 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10059970	A2	19980303	JP 1996-233676	19960816
EP 826687	A1	19980304	EP 1997-306267	19970815
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 6051569	A	20000418	US 1997-911937	19970815

PRIORITY APPLN. INFO.: JP 1996-233676 A 19960816
OTHER SOURCE(S): MARPAT 128:243874
GI



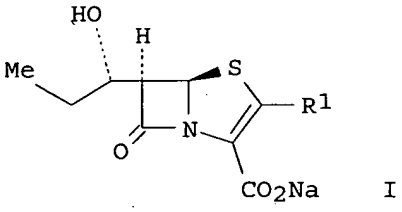
I

AB Carbapenems I [R1 = H, (un)substituted aryl, (un)substituted heterocyclyl; R2 = H, protecting group; R3 = Me, Et; R4 = H] or their pharmacol. acceptable salts, useful as bactericides for methicillin-resistant Staphylococcus aureus (MRSA), etc., are prepared Protected carbapenems I [R1 = (un)substituted alkyl, (un)substituted alkenyl, (un)substituted aryl, (un)substituted heterocyclyl, etc.; R2 = protecting group; R3 = same as above; R4 = H, protecting group] are also claimed. I (R1 = Ph, R2 = allyl, R3 = Et, R4 = H) was treated with Na 2-ethylhexanoate, Ph3P, and (Ph3P)4Pd at room temperature for 15 min in THF to give 22% I (R1 = Ph, R2 = R4 = H, R3 = Et), which had an MIC of 12.5 μg/mL against MRSA.

L24 ANSWER 45 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:109982 HCAPLUS
DOCUMENT NUMBER: 128:200224
TITLE: Reversed micellar mediated luminol chemiluminescence detection of iron(II, III) combined with online solvent extraction using 8-quinolinol
AUTHOR(S): Kyaw, Theingi; Fujiwara, Terufumi; **Inoue, Hidekazu**; Okamoto, Yasuaki; Kumamaru, Takahiro
CORPORATE SOURCE: Department of Chemistry, Faculty of Science, Hiroshima University, Higashi-Hiroshima, 739, Japan
SOURCE: Analytical Sciences (1998), 14(1), 203-207
CODEN: ANSCEN; ISSN: 0910-6340
PUBLISHER: Japan Society for Analytical Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An enhancement of the chemiluminescence (CL) emission, observed when the Fe(III) complex of 8-quinolinol (oxine), Fe(oxine)3, was mixed with a reversed micellar solution of cetyltrimethylammonium chloride in chloroform-cyclohexane (6:5 volume/volume)-H2O (1.0M NaOH) containing luminol and H2O2, was studied to develop a method for Fe(III) determination based on the direct coupling of online solvent extraction with a reversed micellar-mediated CL reaction in a reverse-flow injection system using a microporous Teflon membrane filter for phase separation In the CL process, uptake of the complex by reverse micelles and its subsequent decomposition occurs easily, followed by an Fe(III)-catalyzed luminol reaction. In the online process, Fe(III) was extracted from an aqueous solution into CHCl3 via complex formation with oxine. Upon mixing the reversed micellar luminol solution with the extract stream in a flow cell of a CL monitor, the produced CL signal was measured. A detection limit of 5 ng cm-3 Fe(III) and a linear calibration graph was obtained in the concentration range of 10-500 ng cm-3 Fe(III). In a sample solution containing Fe(II) and Fe(III), total Fe, Fe(II)+Fe(III), was measured as the peak height in the presence of the H2O2 used to oxidize Fe(II) to Fe(III) prior to solvent extraction, while only Fe(III) could be determined in the absence of H2O2.
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 46 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:436027 HCAPLUS
DOCUMENT NUMBER: 127:65612
TITLE: 5,6-cis-Penems: Broad-Spectrum Anti-Methicillin-Resistant Staphylococcus aureus β-Lactam Antibiotics
AUTHOR(S): Ishiguro, Masaji; Tanaka, Rie; Namikawa, Koushi; Nasu, Takaaki; **Inoue, Hidekazu**; Nakatsuka, Takashi; Oyama, Yoshiaki; Imajo, Seiichi
CORPORATE SOURCE: Suntory Institute for Bioorganic Research, Mishima, 618, Japan
SOURCE: Journal of Medicinal Chemistry (1997), 40(14), 2126-2132
CODEN: JMCMAR; ISSN: 0022-2623
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



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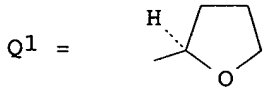
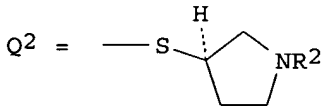


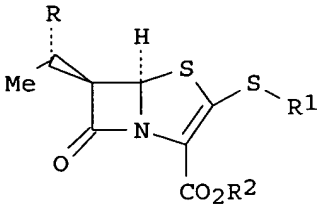


AB 5,6-Cis-penem derivs. (I) (R1 = Ph, Q1, Q2; R2 = H, CH2COPh) have been synthesized and evaluated as anti-MRSA antibiotics. The cis-penems I (R1 = Q2, R2 = H, CH2COPh) showed potent activities against not only MRSA but also a wide variety of bacteria including β-lactamase-producing microorganisms. These compds. were designed to have high affinity to the penicillin-binding protein 2a of MRSA and to form stable acyl intermediates with β-lactamases by blocking the deacylating water mol.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 47 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:433573 HCAPLUS
DOCUMENT NUMBER: 127:50472
TITLE: Preparation of penem derivatives for use as antimicrobial agents
INVENTOR(S): Ishiguro, Masaji; Namikawa, Koshi; Nakatsuka, Takashi; Matsuki, Shinsuke; Tanaka, Rie; **Inoue, Hidekazu**
PATENT ASSIGNEE(S): Suntory Limited, Japan
SOURCE: Eur. Pat. Appl., 79 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 774465	A1	19970521	EP 1996-308409	19961120
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09202789	A2	19970805	JP 1996-233675	19960816
PRIORITY APPLN. INFO.:			JP 1995-323508 A	19951120
			JP 1996-233675 A	19960816

OTHER SOURCE(S): MARPAT 127:50472
GI



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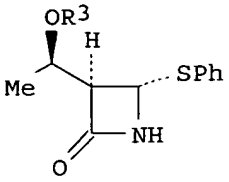

II

AB Penem derivs. I (R = OH, F; R1 = H, alkyl, alkenyl, arylalkyl, aryl, acyl, heterocycle; R2 = H, carboxy protecting group) were prepared for use as antibacterial agents affective against, inter alia, methicillin-resistant Staphylococcus aureus (MRSA). Thus, I (R = OH, R1 = Me, R2 = H) was prepared starting from azetidinone II (R3 = SiMe2CMe3) and gave a MIC value of 12.5 μg/mL when tested against MRSA.

L24 ANSWER 48 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:413189 HCAPLUS
DOCUMENT NUMBER: 127:50287
TITLE: Preparation of optically active trans-vinyl sulfide alcohols as intermediates for penems or carbapenems
INVENTOR(S): Sekiuchi, Kazuto; Imoto, Masahiro; Ishiguro, Shoji; Nakatsuka, Takashi; Tanaka, Rie; **Inoue, Hidekazu**
PATENT ASSIGNEE(S): Suntory, Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 10 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 09124590	A2	19970513	JP 1995-283845	19951031
CA 2209102	AA	19970509	CA 1996-2209102	19961030
WO 9716421	A1	19970509	WO 1996-JP3185	19961030
W: AU, CA, KR, NO, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9673386	A1	19970522	AU 1996-73386	19961030
EP 801057	A1	19971015	EP 1996-935505	19961030
EP 801057	B1	20020116		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
AT 212011	E	20020215	AT 1996-935505	19961030
ES 2171731	T3	20020916	ES 1996-935505	19961030

NO 9703010	A	19970827	NO 1997-3010		19970627
NO 307963	B1	20000626			
US 6049009	A	20000411	US 1997-860563		19970630
AU 772225	B2	20040422	AU 2000-72382		20001219
PRIORITY APPLN. INFO.:			JP 1995-283845	A	19951031
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			WO 1996-JP3185	W	19961030

OTHER SOURCE(S): CASREACT 127:50287; MARPAT 127:50287
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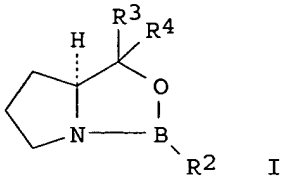

I

AB (R,E)-MeCH(OH)CH:CHSR1 [(R,E)-I; R1 = alkyl, aryl] are prepared by reduction of (E)-MeCOCH:CHSR1 [(E)-II; R1 = same as above] using borane agents in the presence of optically active oxazaborolidines III (R2 = H, alkyl, aryl, aralkyl; R3, R4 = alkyl, aryl, aralkyl) and reduction-controlling additives. Thus, (E)-II (R1 = Ph) was reduced by BH3.Me2S in the presence of III (R2 = Me, R3 = R4 = Ph), mol. sieve 4A, and Me2S in toluene under ice-cooling for 2 h to give 70% (R,E)-I (R1 = Ph) with 90% ee.

L24 ANSWER 49 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:216210 HCAPLUS
DOCUMENT NUMBER: 126:305522
TITLE: Studies on novel and chiral 1,4-dihydropyridines. V. Hantzsch-type 1,4-dihydropyridines having a chiral sulfinyl group: syntheses, structures, and biological activity as a calcium channel antagonist
AUTHOR(S): Miyashita, Kazuyuki; Nishimoto, Masahiro; Ishino, Tetsuya; **Murafuji, Hidenobu**; Obika, Satoshi; Muraoka, Osamu; Imanishi, Takeshi
CORPORATE SOURCE: Fac. Pharmaceutical Sciences, Osaka Univ., Suita, 565, Japan
SOURCE: Tetrahedron (1997), 53(12), 4279-4290
CODEN: TETRAB; ISSN: 0040-4020
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 4-Aryl and 4-Me substituted Hantzsch-type 1,4-dihydropyridines having a chiral sulfinyl group as an electron-withdrawing group were synthesized in an optically active form from β-ketosulfoxides via two routes. The relationship between calcium channel antagonist activity and the structures of the 4-aryl derivs. was also studied.
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 50 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:734680 HCAPLUS
DOCUMENT NUMBER: 126:103987
TITLE: Effect of a neighboring oxygenated substituent on asymmetric reduction with Hantzsch-type 1,4-dihydropyridines having a chiral sulfinyl group

AUTHOR(S): Miyashita, Kazuyuki; Nishimoto, Masahiro; **Murafuji, Hidenobu**; Murakami, Asuka; Obika, Satoshi; In, Yasuko; Ishida, Toshimasa; Imanishi, Takeshi
CORPORATE SOURCE: Fac. Pharmaceutical Sciences, Osaka Univ., Osaka, 565, Japan
SOURCE: Chemical Communications (Cambridge) (1996), (22), 2535-2536
CODEN: CHCOFS; ISSN: 1359-7345
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Introduction of an oxygen-containing substituent at C-6 of a Hantzsch-type compound having a sulfinyl group at C-5 affects the reduction of ketones with resp. to both reactivity and stereoselectivity.
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 51 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:131835 HCAPLUS
DOCUMENT NUMBER: 124:289201
TITLE: Novel and regioselective lithiation of the unsymmetrical Hantzsch-type 1,4-dihydropyridine by participation of the neighboring sulfinyl group
AUTHOR(S): Myashita, Kazuyuki; Nishimoto, Masahiro; **Murafuji, Hidenobu**; Obika, Satoshi; Imanishi, Takeshi
CORPORATE SOURCE: Faculty of Pharmaceutical Sciences, Osaka Univ., Osaka, 565, Japan
SOURCE: Chemical & Pharmaceutical Bulletin (1996), 44(2), 457-9
CODEN: CPBTAL; ISSN: 0009-2363
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 124:289201
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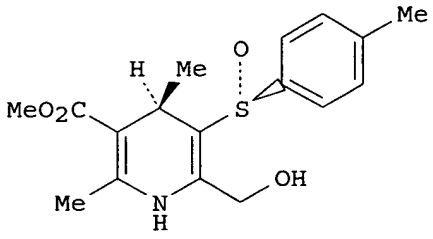

I

AB The C-6 Me group of Me (4R, SS)-2,4,6-trimethyl-5-(p-tolylsulfinyl)-1,4-dihydropyridine-3-carboxylate was found to be regioselectively lithiated by participation of the neighboring sulfinyl group, giving rise to the 6-modified Hantzsch-type compds., e.g., I, by treatment with n-butyllithium and electrophiles.

L24 ANSWER 52 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:605029 HCAPLUS

DOCUMENT NUMBER: 121:205029
TITLE: Copper-assisted substitution reaction for phenylthio group of a 4-(phenylthio)azetidinone derivative
AUTHOR(S): Shimamoto, Tetsuo; **Inoue, Hidekazu**; Yoshida, Takuro; Tanaka, Rie; Nakatsuka, Takashi; Ishiguro, Masaji
CORPORATE SOURCE: Suntory Inst. Biomed. Res., Shimamoto/Osaka, 618, Japan
SOURCE: Tetrahedron Letters (1994), 35(32), 5887-8
CODEN: TELEAY; ISSN: 0040-4039
DOCUMENT TYPE: Journal
LANGUAGE: English
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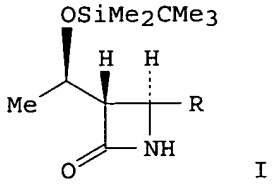

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AB The phenylthio group of 4-(phenylthio)azetidinone I (R = SPh) was readily substituted with copper(I) salts of carboxylates, thiocarboxylates, and copper(I) enolates of malonates and β-ketoesters to give synthetic intermediates I [R = SCOR1, R1 = Me, Ph, 2-tetrahydrofuryl; R = CR2(CO2R3)2, R2 = H, Me, R, R3 = Me, allyl; etc.] for penem and carbapenem antibiotics.

L24 ANSWER 53 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1990:136946 HCAPLUS
DOCUMENT NUMBER: 112:136946
TITLE: Protective immunity against Brugia malayi infective larvae in mice. II. Induction by a T cell-dependent antigen isolated by monoclonal antibody affinity chromatography and SDS-PAGE
AUTHOR(S): Hammerberg, Bruce; Nogami, Sadao; Nakagaki, Kazuhide; **Hayashi, Yashihiro**; Tanaka, Hiroshi
CORPORATE SOURCE: Coll. Vet. Med., North Carolina State Univ., Raleigh, NC, 27606, USA
SOURCE: Journal of Immunology (1989), 143(12), 4201-7
CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A mAb directed against filarial worm secretory/excretory product and reactive with Brugia malayi larval worm surface was used in conjunction with preparative SDS-PAGE to isolate protective antigen (Ag) from exts. of adult B. malayi. The IgM mAb OVH bound to a repeating carbohydrate epitope present in adult, infective, and 4th stage larvae and microfilariae of B. malayi, and on the surface of 4th stage larvae. Ag bearing this epitope were also present in the sea of hosts infected with a variety of helminths, including Brugia, Onchocerca, Dirofilaria, and Paragonimus. Affinity chromatog. of SDS extract of adult Brugia, using mAb OVH immobilized on agarose beads, isolated several Ag that separated into multiple protein staining bands on SDS-PAGE. In comparing SDS-PAGE-fractionated Ag from the crude SDS extract with fractionated mAb OVH-isolated Ag for the ability to protect BALB/c mice from challenge with

B. malayi-infective larvae, it was found that of the mAb OVH-isolated Ag only those at a mol. mass of 26-32 kDa were protective while the original SDS extract yielded protective Ag at the following mol. mass: >200, 170-200, 40-44, 33-36, 23-28, 20-22, and 17-19 kDa. Although Ag isolated by mAb OVH were highly protective, they failed to induce high antibody levels against the immunogen or SDS exts. compared to crude SDS extract immunized mouse sera, as determined by immunoblot and ELISA. Transfer of nylon wool nonadherent T cells from BALB/c mice immunized with the 26-28-kDa fraction of mAb OVH-isolated Ag to naive mice just before challenge with infective larvae of B. malayi resulted in a 70% reduction in larvae recovered 14 days after challenge.

L24 ANSWER 54 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1988:464278 HCAPLUS
DOCUMENT NUMBER: 109:64278
TITLE: Electrophotographic photoreceptor with barrier layer for improved memory effect
INVENTOR(S): Sato, Tsutomu; **Inoue, Hidekazu**
PATENT ASSIGNEE(S): Ricoh Co., Ltd., Japan
SOURCE: Jpn. Tokkyo Koho, 4 pp.
CODEN: JAXXAD
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 63008460	B4	19880223	JP 1977-156934	19771226
JP 54088127	A2	19790713		

PRIORITY APPLN. INFO.: JP 1977-156934 A 19771226

AB An electrophotog. photoreceptor with improved recording ability is comprised of (1) a conductive support, (2) an interlayer as a barrier layer consisting of ≥1 compound selected from polybutadiene, polypropylene, poly(vinyl pyridine), poly(Me methacrylate), polyester and poly(vinyl alc.), and ≥1 compound selected from Mn acetate, CuCl2, phosphomolybdate or its Na salt or its ammonium salt, benzoquinone, and naphthoquinone, and (3) a photoconductive layer consisting of ZnO, CdS or TiO2. The copying method involves: pos. or neg. charging the photoreceptor by corona discharge; imagewise exposing (otherwise nonimage part having zero charges); contacting pos. charges on the image part; and repeating pos. charging, developing and transferring to make ≥2 copies from 1 original copy. The electrophotog. photoreceptor shows excellent memory effect.

L24 ANSWER 55 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1986:159562 HCAPLUS
DOCUMENT NUMBER: 104:159562
TITLE: Electrical insulation-retaining type electrophotographic process
INVENTOR(S): **Inoue, Hidekazu**; Shimizu, Isamu; Nishio, Yoshihiro
PATENT ASSIGNEE(S): Fuji Photo Film Co., Ltd., Japan
SOURCE: Jpn. Tokkyo Koho, 11 pp.
CODEN: JAXXAD
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 60044657	B4	19851004	JP 1977-16710	19770218
JP 53102037	A2	19780906		

PRIORITY APPLN. INFO.: JP 1977-16710 A 19770218

AB The claimed electrophotog. printing process includes the following steps: (1) heat-treatment and neg. charging of an elec. insulating property-retaining electrophotog. plate having a conductive support, which is capable of forming a barrier layer when charged neg., and a photoconductor layer composed of poly(vinylcarbazole), an electron acceptor substance and an electron donor type dye former; (2) imagewise exposure of the electrophotog. plate during or after the exposure; (3) pos. charging of the plate; (4) development of the resultant electrostatic latent images; (5) transfer of the toner images onto a receptor; and (6) repeating of steps (3)-(5) to obtain multiple copies.

L24 ANSWER 56 OF 56 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1975:600255 HCAPLUS
DOCUMENT NUMBER: 83:200255
TITLE: Photochromic photosensitive composition
INVENTOR(S): **Inoue, Hidekazu**; Shimizu, Isamu; Kobayashi, Hajime
PATENT ASSIGNEE(S): Canon K. K., Japan
SOURCE: Jpn. Tokkyo Koho, 5 pp.
CODEN: JAXXAD
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 50010273	B4	19750419	JP 1969-86618	19691029

PRIORITY APPLN. INFO.: JP 1969-86618 19691029

AB A photochromic composition which produces coloration efficiently and does not show fatigue is prepared by dispersing a nonpolar photochromic substance in a polar medium with the use of a surfactant. The photochromic composition can be used in image recording, photomasking, copying and microphotog. Spiropyrans are especially preferred as the photochromic substance. Thus, the mixture of 1,3,3-trimethyl-6'-nitrospiro[indoline-2,2'-benzopyran] 50 and TAMDO 10 (a surfactant) 500 mg was added to a solution containing poly(vinyl alc.) 2 g in H2O 2/ ml, coated on a Ni-plated plate, dried to give a 30-50 μ layer, exposed to a 500-W high-pressure Hg lamp for 1 min at 20 cm using a glass light filter (UVD-25) to produce a coloration having an absorption maximum at 5500 Å and an optical d. of 0.5. The color d. reduced to 0.37 when stored at 22° in a dark place for 24 hr. Exposed to the visible light from a 1-kW W lamp the color was completely bleached in 20 sec.

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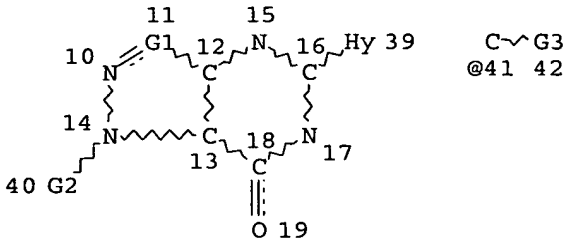

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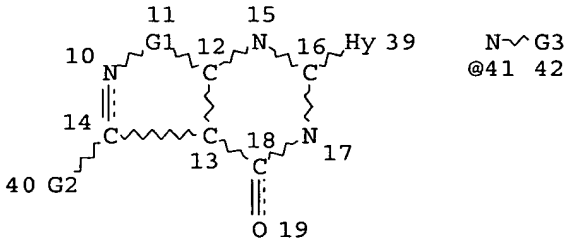

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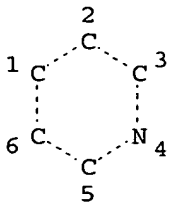

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STEREO ATTRIBUTES: NONE

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L21	14	SEA	FILE=HCAPLUS ABB=ON	PLU=ON	("MURAFUJI H"/AU OR "MURAFUJI HIDENOBU"/AU)
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L25 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:874453 HCAPLUS
DOCUMENT NUMBER: 137:346047
TITLE: The efficacy of alendronate in reducing the risk for vertebral fracture in Japanese patients with osteoporosis: a randomized, double-blind, active-controlled, double-dummy trial
AUTHOR(S): Kushida, Kazuhiro; Shiraki, Masataka; Nakamura, Toshitaka; **Kishimoto, Hideaki**; Morii, Hirotoshi; Yamamoto, Kichizo; Kaneda, Kiyoshi; Fukunaga, Masao; Inoue, Tetsuro; Nakashima, Mituyoshi; Orimo, Hajime; Ross, Philip D.; Thompson, Desmond E.; Sato, K.; Tanba, J.; Fukuchi, M.; Ichikawa, T.; Kawasaki, S.; Oguma, T.; Hyakutake, S.; Goto, S.; Moriya, H.; Saotome, K.; Masuda, T.; Kim, K.; Morioka, K.; Koyama, S.; Matsubayashi, T.; Yoshizawa, H.; Baba, H.; Imura, S.; Maezawa, Y.; Narita, S.; Arinaga, M.; Kido, M.; Matsuzaki, A.; Nakamura, H.; Kikuchi, S.; Sato, M.; Sugimoto, T.; Fukumoto, Y.; Hashimoto, T.; Satoh, T.; Nakamura, T.; Nakashima, M.; Fujiwara, T.; Hoshino, H.; Inoue, T.; Kushida, K.; Ito, A.; Ohnishi, T.; Okamura, H.; Katano, H.; Komatu, T.; Harada, S.; Kaneda, K.; Taneichi, H.; Maruo, S.; Yoh, K.; Jujii, K.; Sai, S.; Yamashita, K.; Mori, S.; Norimatsu, H.; Sakou, T.; Yone, K.; Morimoto, K.; Funato, T.; Fukunaga, M.; Suzuki, K.; Chihara, K.; Fujita, K.; Mizuno, K.; Sugimoti, T.; Yamamoto, H.; Takahashi, T.; Takemasa, R.; Mashiko, M.; **Hayashi, Y.**; Fukuyama, S.; Ohya, T.; Saita, M.; Ishii, Y.; Ohtani, K.; Sekioka, Y.; Koh, T.; Iwaba, Y.; Kusakabe, T.; Konishi, J.; Nakamura, T.; Sigeno, C.; Tsuboyama, T.; Nawata, H.; Takayanagi, R.; Hagiwara, Y.; Sudou, A.; U&chida, A.; Hieda, H.; Kiriyama, T.; Mameya, G.; Seto, M.; Iwasaki, K.; Kiriyama, K.; Nagataki, S.;

Tomonaga, T.; Yokoyama, N.; Matsui, N.; Iwata, H.; Ishiguro, N.; Shimizu, S.; Nagata, I.; Yamamoto, M.; Azuma, Y.; Takizawa, H.; Harada, A.; Kobayashi, M.; Suenaga, N.; Takahahi, H.; Seki, T.; Takahashi, H. E.; Horiuchi, T.; Masuyama, H.; Tojima, T.; Fujiwara, M.; Kasai, R.; Naka, K.; Kuragami, C.; Ohya, T.; Kakizeo, M.; Harada, Y.; Inoue, H.; Kadoya, Y.; Miki, T.; Morii, H.; Nishizawa, Y.; Yamano, Y.; Hashimoto, J.; Nakase, T.; Ochi, T.; Shiraki, M.; Sadamatsu, T.; Itabashi, A.; Katayama, S.; Hasegawa, Y.; Sakamoto, H.; Shiotani, A.; Okamoto, S.; Mochizuki, T.; Yamazaki, Y.; Ishii, S.; Takada, J.; Fukuda, S.; Ohno, K.; Mieno, T.; Hashizume, K.; Kobayashi, S.; Suzuki, S.; Takaoka, K.; Fujimaki, E.; Sakamoto, K.; Konishi, N.; Suda, A.; Kadowaki, T.; Mizushima, M.; Tsutsumi, M.; Miki, H.; Ohno, K.; Yagi, T.; Keijinkai, Teine; Koizumi, Y.; Kokubun, S.; Takamatsu, K.; Tanaka, Y.; Hosoi, T.; Itou, H.; Karube, S.; Orimo, H.; Yamamoto, S.; Yoshida, K.; Nakano, T.; Miyazaki, S.; Takahashi, H.; Hagino, H.; Kishimoto, H.; Yamamoto, K.; Tsuji, H.; Matsubara, T.; Abe, A.; Ueno, T.; Suzuki, K.; Nakamura, T.; Ibaraki, K.; Ikata, T.; Kashiwaguchi, S.; Takada, S.; Hosoi, T.; Ouchi, Y.; Tamaki, T.; Kotake, H.; Akamatsu, N.; Nakajima, I.; Onaya, T.

CORPORATE SOURCE: Hamamatsu University School of Medicine, Hamamatsu, Japan

SOURCE: Current Therapeutic Research (2002), 63(9), 606-620
CODEN: CTCEA9; ISSN: 0011-393X

PUBLISHER: Excerpta Medica, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Alendronate, a potent antiresorptive agent, effectively reduces fracture risk. Alendronate increases bone mineral d. (BMD) and decreases bone turnover markers to a similar extent in white and Asian people, including Japanese. However, no large trials of this drug have been conducted specifically in the Japanese population. This study examined the antifracture efficacy of alendronate in Japanese patients. We conducted a 2-yr, multicenter, randomized, double-blind, active-controlled, double-dummy trial of women and men with osteoporosis in Japan, with radiog. diagnosed vertebral fracture being the primary end point. Patients were randomized to receive alendronate 5 mg/d or alfacalcidol 1 μg/d. A total of 365 patients (349 women, 16 men; mean age, 73 yr) were enrolled in the study. At the end of 24 mo, spinal BMD was significantly increased vs. baseline by a mean of 6.9% in the alendronate-treated group ($P < 0.001$) and 1.5% in the alfacalcidol-treated group; the median increases in BMD at 24 mo vs. baseline were 8.3% and 1.4%, resp. The incidence of vertebral fracture >6 mo after randomization (the primary end point) was significantly reduced by 66% (relative risk [RR], 0.34; 95% CI, 0.15-0.74) in the alendronate group (4.3% vs 12.7% incidence). When all fractures during the 24 mo were considered, the incidence of multiple (≥2) vertebral fractures also was reduced significantly by 67% (RR, 0.33; 95% CI, 0.11-0.96; 2.4% vs 7.3% incidence). The difference in overall incidence (≥1 vertebral fracture during all 24 mo) was not significant (12.2% vs 16.70), implying that risk redns. (relative to the active control) improved after 6 mo. This is consistent with bone biol. theory, which predicts that several months are required to refill existing resorption sites and increase bone strength more than the active control. Thus, theory predicts that several months would be required to increase bone strength and reduce fracture risk relative to the active control. This study was limited to Japanese

participants, but the findings are consistent with results reported from similar studies among white people. Furthermore, because this was an active-controlled trial, it may have underestimated the antifracture efficacy of alendronate relative to a true placebo. The effectiveness of alendronate in reducing the risk of radiog. defined vertebral fracture in Japanese women and men with osteoporosis is similar to that reported previously in white people. As a consequence, the benefits of alendronate in reducing vertebral and hip fractures can be expected to be substantial and early (beginning at month 6).

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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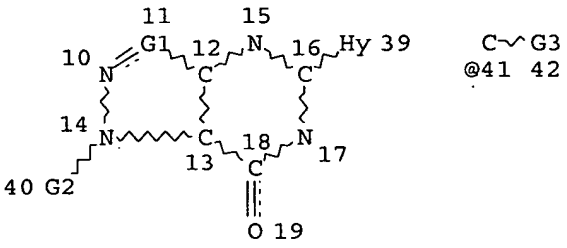

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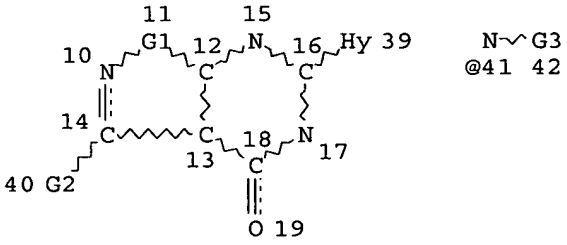

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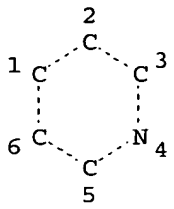

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L23 728 SEA FILE=HCAPLUS ABB=ON PLU=ON "HAYASHI Y"/AU
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L25 1 SEA FILE=HCAPLUS ABB=ON PLU=ON (L20 AND L23) NOT (L18 OR L24)
L28 3 SEA FILE=HCAPLUS ABB=ON PLU=ON ((L20 OR L23) AND (PDE OR PHOSPHODIESTERASE OR PYRIDINYLPYRA?)) NOT (L18 OR L24 OR L25)

=> d ibib abs hitstr l28 1-3

L28 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:575531 HCAPLUS
DOCUMENT NUMBER: 135:267593
TITLE: Role of adenosine and P2 receptors in the penile tumescence in anesthetized dogs
AUTHOR(S): Noto, T.; **Inoue, H.**; Mochida, H.; Kikkawa, K.
CORPORATE SOURCE: Discovery Research Laboratory, Tanabe Seiyaku Co., Ltd., Kawagishi, Toda, Saitama, 335-8505, Japan
SOURCE: European Journal of Pharmacology (2001), 425(1), 51-55
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors studied the role of adenosine and P2 receptors in the pelvic nerve stimulation-induced penile tumescence in anesthetized dogs. A local intracavernous injection of adenosine induced the tumescence, which was abolished by intracavernous 8-(p-sulfophenyl)theophylline (8-SPT), an

unspecific adenosine receptor antagonist, and by 4-(2-[7-amino-2-(2-furyl)[1,2,4]triazolo[2,3-a][1,3,5]triazin-5-yl amino]ethyl)phenol (ZM241385), an adenosine A2A receptor antagonist. ATP also induced the tumescence, which was diminished by 8-SPT, but not by reactive blue-2, a P2 receptor antagonist. Neither intracavernous β,γ-meATP nor ADPβS, P2X and P2Y receptor agonists, induced tumescence. NG-nitro-L-arginine (l-NAME), a nitric oxide synthase inhibitor, and T-1032, a **phosphodiesterase** type V inhibitor, had no effects on the tumescence induced by adenosine. Addnl., 8-SPT and reactive blue-2 had no effects on the tumescence induced by pelvic nerve stimulation. These results show that although exogenous adenosine and ATP induce tumescence, neither the adenosine nor the P2 receptor is involved in the tumescence induced by pelvic nerve stimulation in anesthetized dogs.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:467698 HCAPLUS

DOCUMENT NUMBER: 135:298499

TITLE: T-1032, a novel specific **phosphodiesterase** type 5 inhibitor, increases venous compliance in anesthetized rats

AUTHOR(S): **Inoue, H.**; Yano, K.; Ikeo, T.; Noto, T.; Kikkawa, K.

CORPORATE SOURCE: Discovery Research Laboratory, Tanabe Seiyaku Co., Ltd., Toda, Saitama, Kawagishi, 335-8505, Japan

SOURCE: European Journal of Pharmacology (2001), 422(1-3), 109-114
CODEN: EJPHAZ; ISSN: 0014-2999

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Nitric oxide (NO) donors including organic nitrates dilate capacitance vessels. As inhibition of **phosphodiesterase** type 5 results in the accumulation of guanosine 3'-5'-cyclic monophosphate (cGMP), specific **phosphodiesterase** type 5 inhibitors are expected to have a vasodilator property similar to that of NO donors. To test this hypothesis, we examined the effect of methyl-2-(4-aminophenyl)-1,2-dihydro-1-oxo-7-(2-pyridinylmethoxy)-4-(3,4,5-trimethoxyphenyl)-3-isoquinoline carboxylate sulfate (T-1032), a novel specific **phosphodiesterase** type 5 inhibitor, on mean arterial pressure and mean circulatory filling pressure (an index of venodilation) compared with that of nitroglycerin and diltiazem in mecamylamine- and noradrenaline-treated anesthetized rats. I.v. infusion of T-1032 (0.1, 1, 10 μg/kg/min) dose-dependently decreased mean arterial pressure (-3.8±0.3%, -9.1±0.8%, -16.8±1.5% at doses of 0.1, 1 and 10 μg/kg/min, resp.) and mean circulatory filling pressure (-6.1±0.9%, -12.5±0.7%, -18.6±3.0% at doses of 0.1, 1 and 10 μg/kg/min, resp.). The mean circulatory filling pressure-mean arterial pressure relationship revealed that T-1032 had a selective action on the mean circulatory filling pressure compared with diltiazem (10, 100 μg/kg/min) and a similar or more selective effect than nitroglycerin (0.3, 3 and 30 μg/kg/min). In the next study, we calculated venous compliance and unstressed volume from the mean circulatory filling pressure-volume relationship. I.v. infusion of T-1032 (3 μg/kg/min) increased venous compliance (3.35±0.40 in T-1032 vs. 2.31±0.15 mL/kg/mm Hg in vehicle, $P<0.05$) without changing the unstressed volume (37.2±2.80 in T-1032 vs. 42.6±2.37 mL/kg in vehicle, $P>0.05$). It was concluded that T-1032 increased venous capacitance by increasing venous compliance, and that this selective **phosphodiesterase** type 5 inhibitor appeared to have a different

vasodilator action from that of an NO donor and a Ca2+ channel antagonist in that it had a selective action on the mean circulatory filling pressure.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:554787 HCAPLUS
DOCUMENT NUMBER: 121:154787
TITLE: Involvement of cyclic AMP-generating systems in cortical epileptic activity.
AUTHOR(S): Hattori, Y.; Moriwaki, A.; **Hayashi, Y.**; Hori, Y.
CORPORATE SOURCE: Department Physiology, Okayama University Medical School, Shikata, 700, Japan
SOURCE: Neurosciences (Okayama, Japan) (1994), 20(SUPPL.), P157-P160
CODEN: NUOCDO; ISSN: 0388-7448
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB The cAMP accumulation in slices incubated with adenosine and its stable analog 2-chloroadenosine was investigated in four different regions of rat cerebral cortex which exhibited electrog. and behavioral epileptic activities following unilateral injection of cobalt chloride solution into the sensorimotor cortex. Adenosine and 2-chloroadenosine elicited cAMP accumulation and the elicitation was strongly inhibited by the adenosine antagonist 8-phenyltheophylline. The cAMP accumulation was increased in the primary cortical region of cobalt-induced epilepsy, but not in the other cortical regions. The increase in cAMP accumulation was detected regardless of the presence or absence of the adenosine uptake inhibitor, **phosphodiesterase** inhibitor, and adenosine deaminase. The cAMP accumulation, which was at the maximal level 17-19 days after treatment by cobalt, was similar in time course to the electrog. spike activity. These results suggest that adenosine-sensitive generation of cAMP is closely associated with central mechanism of cobalt-induced epilepsy.

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     (FILE 'HCAPLUS' ENTERED AT 14:29:12 ON 16 MAR 2006)
               DEL HIS Y

     FILE 'REGISTRY' ENTERED AT 14:48:55 ON 16 MAR 2006
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L3             STR L1
L4             STR L3
L5          41 SEA SSS SAM L4
L6         844 SEA SSS FUL L4
L7             STR L1
L8           0 SEA SUB=L6 SSS FUL L7
               D L4
L9             STR L7
L10          9 SEA SUB=L6 SSS FUL L9
L11            STR L4
L12            STR L11
L13            STR L12
L14          1 SEA SSS SAM L12 OR L13
               D SCAN
L15         46 SEA SSS FUL L12 OR L13
L16            STR
L17         44 SEA SUB=L15 SSS FUL L16

     FILE 'HCAPLUS' ENTERED AT 15:05:10 ON 16 MAR 2006
L18          4 SEA ABB=ON  PLU=ON  L17

     FILE 'HCAPLUS' ENTERED AT 15:05:31 ON 16 MAR 2006
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               D IBIB ABS HITSTR L18 1-4
               E INOUE H/AU
               E INOUE HIDEKAZU/AU
L19         47 SEA ABB=ON  PLU=ON  "INOUE HIDEKAZU"/AU OR "INOUE HIDEKAZU C O-
               DAINIPPON S"/AU
L20        608 SEA ABB=ON  PLU=ON  INOUE H/AU
               E MURAFUJI H/AU
L21         14 SEA ABB=ON  PLU=ON  ("MURAFUJI H"/AU OR "MURAFUJI HIDENOBU"/AU)

               E HAYASHI Y/AU
L22          1 SEA ABB=ON  PLU=ON  "HAYASHI YASHIHIRO"/AU
L23        728 SEA ABB=ON  PLU=ON  "HAYASHI Y"/AU
L24         56 SEA ABB=ON  PLU=ON  (L19 OR L21 OR L22) NOT L18
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L25          1 SEA ABB=ON  PLU=ON  (L20 AND L23) NOT (L18 OR L24)
               D STAT QUE L25
               D IBIB ABS HITSTR L25 1
L26        141 SEA ABB=ON  PLU=ON  ((L20 OR L23) AND (PDE OR PHOSPHODIESTERASE
                OR PYRIDINYLPYRA? OR INHIBIT?)) NOT (L18 OR L24 OR L25)
L27          0 SEA ABB=ON  PLU=ON  ((L20 OR L23) AND ((PDE OR PHOSPHODIESTERAS
               E OR INHIBIT?)(L)PYRIDINYLPYRA?)) NOT (L18 OR L24 OR L25)
L28          3 SEA ABB=ON  PLU=ON  ((L20 OR L23) AND (PDE OR PHOSPHODIESTERASE
                OR PYRIDINYLPYRA?)) NOT (L18 OR L24 OR L25)
               D SCAN
               D STAT QUE
               D IBIB ABS HITSTR L28 1-3
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FILE REGISTRY
Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

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* effective March 20, 2005. A new display format, IDERL, is now *
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FILE LAST UPDATED: 15 Mar 2006 (20060315/ED)

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 NEWS  4  JAN 13   IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS  5  JAN 13   New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                   INPADOC
 NEWS  6  JAN 17   Pre-1988 INPI data added to MARPAT
 NEWS  7  JAN 17   IPC 8 in the WPI family of databases including WPIFV
 NEWS  8  JAN 30   Saved answer limit increased
 NEWS  9  FEB 21   STN AnaVist, Version 1.1, lets you share your STN AnaVist
                   visualization results
 NEWS 10  FEB 22   The IPC thesaurus added to additional patent databases on STN
 NEWS 11  FEB 22   Updates in EPFULL; IPC 8 enhancements added
 NEWS 12  FEB 27   New STN AnaVist pricing effective March 1, 2006
 NEWS 13  FEB 28   MEDLINE/LMEDLINE reload improves functionality
 NEWS 14  FEB 28   TOXCENTER reloaded with enhancements
 NEWS 15  FEB 28   REGISTRY/ZREGISTRY enhanced with more experimental spectral
                   property data
 NEWS 16  MAR 01   INSPEC reloaded and enhanced
 NEWS 17  MAR 03   Updates in PATDPA; addition of IPC 8 data without attributes
 NEWS 18  MAR 08   X.25 communication option no longer available after June 2006
 NEWS 19  MAR 22   EMBASE is now updated on a daily basis
 NEWS 20  APR 03   New IPC 8 fields and IPC thesaurus added to PATDPAFULL
 NEWS 21  APR 03   Bibliographic data updates resume; new IPC 8 fields and IPC
                   thesaurus added in PCTFULL
 NEWS 22  APR 04   STN AnaVist $500 visualization usage credit offered
 NEWS 23  APR 12   LINSPEC, learning database for INSPEC, reloaded and enhanced
 NEWS 24  APR 12   Improved structure highlighting in FQHIT and QHIT display
                   in MARPAT
 NEWS 25  APR 12   Derwent World Patents Index to be reloaded and enhanced during
                   second quarter; strategies may be affected

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